Paraquat

Unacceptable health risks for users

3rd Edition

BD
Berne Declaration
Déclaration de Berne
Erklärung von Bern

PESTICIDE ACTION NETWORK UK

EMPOWERING PEOPLE FOR CHANGE
PANAP
PESTICIDE ACTION NETWORK ASIA & THE PACIFIC

Written by Lars Neumeister (MSc., M.Eng.) and Richard Isenring (MSc.)

March 2011

Berne Declaration, Quellenstrasse 25, P.O.Box, CH8031 Zürich Switzerland Phone: +41 44 277 70 00, Email: food@evb.ch, Web: www.evb.ch

PAN UK, Pesticide Action Network UK, Development House, 56-64 Leonard Street, London EC2A 4JX, UK Phone: +44 (0)20 7065 0905, Email: admin@pan-uk.org, Web: www.pan-uk.org

PAN Asia and the Pacific, P.O. Box 1170, Penang, 10850 Malaysia, Tel: 604-657 0271 or 604-656 0381 Fax: 604-6583960, E-mail: panap@panap.net Web: www.panap.net

Please visit www.stop-paraquat.net for more and updated information about paraquat.

How to cite: Neumeister L & Isenring R (2011): Paraquat. Unacceptable health risks for users. 3rd Edition. Berne Declaration, Pesticide Action Network UK, PAN Asia and the Pacific.

Table of Content

1. Preface by the Publishers

In 2006, the last edition of this report was published. In these last 5 years a lot has happened. The claim of Sweden that '*(...) paraquat is the substance most dangerous to health — in terms of acute toxicity — ever included in Annex I to Directive 91/414 (...)*' eventually lead to an EU–wide ban of paraquat including the requirement for an export notification similar to the international PIC procedure.

On an international level, several UN Organisations admitted that all previous attempts to manage chemicals, including pesticides safely have failed. Literally, UNEP's SAICM[1] document states that: '*existing international policy framework (...) is not completely adequate (....). Coherence and synergies between existing institutions and processes are not completely developed (...). There is often limited or no information on many chemicals currently in use (...). Many countries lack the capacity to manage chemicals soundly at the national, subregional, regional and global levels (...)*' (UNEP 2006 pg. 12.)

In conclusion, UNEP proposes: '*Base national decisions on highly toxic pesticides on an evaluation of their intrinsic hazards and anticipated local exposure to them*' (UNEP 2006 pg. 44). This presents a paradigm shift. 'Traditional' (industry–friendly) risk management assumed that all pesticides could be used safely, as long as users oblige to the instructions. With time, the international policy arena recognized that 'Safe/Proper Use' of pesticides under the prevailing socio–economic and political conditions in developing countries and countries in transition is an illusion. And they are right: there are millions of very poor farmers and plantation workers–which resources must be made available to provide the necessary education, improve working condtions, and strenghthen controls in agriculture?

While supporting the interests of the pesticide industry and plantation companies–and proposing training plus voluntary measures–tens of thousand people have suffered from highly toxic pesticides such as paraquat. A shift towards more restrictive measures is the right thing to do.

There is some more good news. After thousands of suicides which were committed with paraquat in Sri Lanka, the government there eventually banned all uses of paraquat. Burkina Faso has proposed to add 'Gramoxone Super' to Annex III of the Rotterdam Convention, because of numerous occupational poisonings caused by this highly toxic paraquat formulation. Dole's and Chiquita's production is paraquat–free and many certification organisations and large food processors and/or retailers have eliminated paraquat from their supply chain and production systems.

While some progress can be seen, this revised report reveals that adverse effects of paraquat continue to exist. In South Korea, 1,200 to 1,400 people die annually from ingesting paraquat and accidental ingestion is still common accross the world. This report shows that the herbicide paraquat causes daily suffering to a very large number of farmers, workers and their families. Problems resulting from paraquat exposure are found around the world: from the United States to Japan and from Costa Rica to Malaysia. The injuries suffered are debilitating and sometimes fatal. Associated chronic health problems are now being identified.

This report also shows that many basic conditions have not changed: In developing countries in particular, paraquat is widely used under high–risk conditions. Problems of poverty are exacerbated by the exposure to hazardous chemicalss, as users have no means to protect themselves. Personal protective equipment is largely not available; it is costly and impossible to wear in hot working conditions. While education, training and information about alternatives and pesticide risk are urgently needed to avoid pesticide use and poisonings, the basic problem is the use of high-risk chemicals like paraquat under conditions of poverty.

1 UNEP (2006): Strategic Approach to International Chemicals Management. SAICM texts and resolutions of the International Conference on Chemicals Management. United Nations Environment Programme (UNEP). www.saicm.org

Therefore, the Berne Declaration, Pesticide Action Network Asia & the Pacific and Pesticide Action Network UK demand the following from:

Syngenta and other manufactures of paraquat

- to immediately halt the production and marketing of pesticides containing paraquat
- to stop manipulating decision making processes regarding pesticide authorization or restrictions
- to withdraw and not fund misleading information about 'safe' practices of pesticide use

National authorities

- to withdraw all authorizations of products containing paraquat
- to base regulatory decisions on the real–life situation of farmers, plantation workers and others exposed to pesticides

Food and Agricultural Organisation (FAO)

- to provide more support for national authorities to phase out highly hazardous pesticides, like paraquat, and for the reduction of pesticide dependency
- to amend the Code of Conduct on the distribution and use of pesticides to explicitly incorporate the phasing out of highly hazardous pesticides
- to amend the Code of Conduct on the distribution and use of pesticides in a way that it respects the activities regarding pesticides suggested by SAICM

World Health Organisation (WHO)

- to classify paraquat (dichloride) as a WHO Ia pesticide based on its real mortality rates
- to identify fatality rates for all common pesticides and in various contexts (suicidal, occupational, accidental)

the Conference of the Parties (COP) of the Rotterdam Convention

- to add paraquat to Annex III of the Rotterdam Convention similar to other acutely toxic pesticides which have proven fatal

the food industry, food retailers and label organisations

- to ban the use of paraquat in the supply chain

2. Summary

The highly toxic herbicide paraquat has been used for about 50 years. Among all common ly used herbicides it has by far the highest acute toxicity, and compared to all other pesticides is has the highest mortality rate. Paraquat is currently the deadliest pesticide on the market – which is a result of its very high acute toxicity combined with the absence of an antidote.

In developing countries and countries in transition paraquat is often used without protection. Even when applied with protective equipment, adverse health effects cannot be excluded. In general, paraquat poisonings have have fundamental socio-economic causes, most prominently: poverty, illiteracy, lack of education, and among plantation workers lack of alternative income opportunities. In many countries, legislation on occupational safety and health is weak and/or not enforced. Authorization of pesticides does not respect local conditions and pathways of exposure. Knowledge of farmers how they can reduce pesticide dependency and of farmworkers how risks of using paraquat can be reduced remains low.

From the time when it was first marketed until today, accidental and intentional drinking of paraquat has caused an innumerable loss of lifes. Fatal poisoning at the workplace occurs mostly when paraquat absorption through skin is increased after prolonged contact with undiluted or diluted paraquat solution. Spray mist deposited in the nose may be swallowed and spray in the air can be ingested when workers breathe through the mouth. The level of exposure to paraquat that workers may experience when handling paraquat is high enough to lead to absorption of an amount that can result in acute poisoning. The symptoms of poisonings are often delayed. Damage to the lungs, for example, may not be evident until several days after absorption. Since there is no antidote against paraquat poisoning, the outcome can be fatal and in these cases death mostly results from respiratory failure.

Localised skin damage, eye injury and nose bleed occur frequently among paraquat users, requiring medical treatment that is often not available. Long–term exposure to low doses of paraquat may result in changes in the lung and appears to be connected with chronic bronchitis and shortness of breath. Exposure to paraquat has been associated with an increased risk of developing Parkinson‘s disease.

In some countries paraquat has caused such a high number of suicide victims that is has been banned or severely restricted. All other approaches have failed –neither new formulations nor the industry‘s ‚safe storage‘ campaigns had any success.

Paraquat presents an acute hazard to small mammals, birds, beneficial insects and fish. If groundnesting birds are exposed to spray this can affect their reproduction. Residues of paraquat above the drinking-water limit have been measured in surface waters and in drinking water. The very low degradation rate of paraquat in soil may lead to an accumulation in soil. No–till systems facilitate accumulation of paraquat in the topmost soil layer.

It is now over 25 years that the international community tries – on a voluntary basis – to manage risks associated with the use of pesticides. Twenty years after the first FAO Code of Conduct on the use and distribution of pesticides was established, the Strategic Approach to International Chemicals Management (SAICM) admits that all risk management strategies in developing countries have failed. Eventually, the FAO called for a phasing out of highly hazardous pesticides, and SAICM now asks regulatory bodies to base decisions on the anticipated local exposure.

While the international community is petrified regarding chemical safety, paraquat has been banned regionally. The European Union banned all uses of paraquat and requires export notifications from all Member States. Burkina Faso, one of the countries with the lowest literacy rate worldwide, proposed to add ‘Gramoxone Super’ to Annex III of the Rotterdam Convention, because, the use of this highly toxic pesticide has caused numerous acute poisonings.

All major labels (certification standards) aiming at more environment–friendly and socially fair production have also prohibited paraquat, and numerous retailers and food processors demand that their suppliers do not use paraquat. Paraquat–free agriculture is now implemented in regions all over the world and across all crops.

On the other hand, ‘Safe Use’ campaigns carried out by the pesticide industry have not been able to change the behaviour of a significant proportion of farmers and agricultural workers.

3. Introduction

The use of the herbicide paraquat has been a subject of controversy for several decades, especially regarding the safety of farmers and agricultural workers in developing countries (Madeley 2002a, 2002b; Wesseling et al. 2001a). Both intentional and unintentional poisonings with paraquat, mainly among agricultural workers, farmers and inhabitants of rural areas, have led to serious concern among national health authorities, workers' unions and non-governmental organisations.

Acutely toxic pesticides are used in many countries under inadequate conditions and contribute considerably to ill health and premature deaths, both among agricultural workers and the general public.

This paper presents the findings by experts, national and international organisations on the general causes of human pesticide poisonings, paraquat exposure, health and environmental effects of paraquat, and policies installed aiming at risk reduction. Voluntary production standards regulating pesticides as well as paraquat alternatives are shown.

3. 1 The active substance paraquat

Paraquat was first introduced in Malaysian rubber plantations in 1961 (Calderbank & Farrington 1995). Its use has since become widespread. The largest producing nation is currently China. As a broad-spectrum (or nonselective) herbicide, paraquat kills both broad leaved weeds and grasses. It is used on fruit and plantation crops (banana, cocoa, coffee, oil palm), field crops (maize), in direct seeding (or conservation tillage), in forestry and as defoliant or desiccant to dry crop plants (cotton, pineapple, soy bean, sugar cane, e.g.) (Tomlin 2003). Paraquat has also been used for control of aquatic weeds (Visamara et al. 2000).

Paraquat belongs to the small group of bipyridylium herbicides which are quaternary ammonium salts (known as 'quats'). Paraquat is a contact herbicide, which will affect all exposed green parts of a plant, but it does not move within the plant (non-systemic). Products based on paraquat normally contain the dichloride salt of paraquat cation.

Paraquat is applied before sowing or planting the crop, in pre-emergence application (before planting or before emergency of seedlings) or as a defoliant before harvest (Hall 1995a).

In liquid concentrate form, it is usually diluted by agricultural workers in the field before spraying. To kill weeds, paraquat is applied at rates of 0.28 to 1.12 kg/ha (1/4 to 1 lb per acre); for desiccation it may be used twice (Hall 1995a).

The liquid concentrates of paraquat contain 5% to 44% of the active substance, and also solvent (water) and wetting agents or adjuvants. Granular (solid) formulations are used less frequently (Hall 1995a).

Paraquat is sold under various trade names and a newer list of tradenames has been compiled by PAN Asia & the Pacific (PANAP 2010). The main product line is 'Gramoxone', marketed by Syngenta. In 2007, Syngenta stated, that it has 75% market share[2], but since then this share has probable decreased due to increased generica production esp. by Chinese companies.

4. Causes of human pesticide poisonings

The underlying causes of pesticide poisonings are very complex: socioeconomic factors (poverty, education, debts of individuals, corruption and profit maximization), policy (regulation, enforcement), climate and individual behavior are related and interdependent. In many developing countries[3] most of the predisposing factors occur simultanously: A low level of literacy (e.g. see Figure 1) leads to mis- and overuse of pesticides, because information about proper use and less toxic alternatives are not accessible, there are not enough financial resources to buy protective equipment, while on the government side there are no resources to educate farmers and to enforce legal obligations (for examples see Box 1 and Annex I). Lack of education renders the search for alternative sources of income impossible, and as a result employed plantation workers

2 Speech of Mr. Taylor at Syngenta's General Assembly May 2nd 2007.

3 Throughout the report, the term 'developing countries' includes countries with economies in transition.

Box 1: BANGLADESH: Pesticide poisoning takes its toll

DHAKA, 18 January 2010 (IRIN) - An annual government survey of Bangladesh's health situation has found that pesticide-related poisoning is a leading cause of death, underscoring a major health concern.

The 2009 Health Bulletin (...) recorded 7,438 pesticide-related poisoning deaths at more than 400 hospitals nationwide amongst men and women aged 15-49.

The use of chemicals for growing vegetables was a major factor in the pesticide-related deaths, said Muhammad Abul Faiz, professor of medicine, previously director-general of health services for the government.

„*Farmers apply pesticides on their crops without taking proper protective measures. They expose themselves to highly poisonous pesticides. They inhale substantial amounts of the pesticides they spray to kill insects in their crops*," Faiz told IRIN.

Farmers apply pesticides on their crops without taking proper protective measures. They expose themselves to highly poisonous pesticides. „*Others get poisoned because they do not properly wash their hands and faces after spraying pesticides*," he said.

That is bad news in a country where 75 percent of the civilian labour force - estimated at 56 million - is directly or indirectly engaged in the agriculture sector.

Dangerous recycling

Scientists from the National Institute of Preventive and Social Medicine (NIPSOM) report that many farmers do not dispose of empty pesticide containers after use, instead routinely recycling them. Sometimes the containers are used for storing food items, underscoring the importance of proper recycling and disposal of used containers, they say.

NIPSOM scientists also say people need to be made aware of poisoning caused by recycling and improper disposal of used pesticide containers. They recommend that pesticide dealers ensure that warnings are explicitly written on containers, so they are not used for the storage of any food item.

But this is a challenge, since the country's adult literacy rate is only 56.3 percent, according to government figures.

„*Considering the widespread illiteracy of our farmers, it should be made mandatory for pesticide producers and sellers to print pictures on pesticide containers showing how to use and dispose of them properly after use*," said Mohammad Mahfuzullah, an environmental activist and executive director of the Centre for Sustainable Development (CFSD), a national NGO. (...)

Regulations ignored

Bangladesh's 1985 Pesticide Rules outline stringent procedures for the registration, import, manufacture, sale, packaging and advertisement of pesticides.

But pesticide importers and traders pay scant attention to these regulations, experts say.

Illiterate farmers are also persuaded by unscrupulous traders and various incentive schemes to buy unregistered pesticide formulations that promise to protect crops against pest attacks and disease.

Meanwhile, suppliers continue to sell many chemical substances banned by the government, as well as chemical compounds such as aldrin and endrin, which are classified as „highly hazardous" by the World Health Organisation (WHO).

In addition, many pesticides continue to be sold in the market without names or under false labels, and with no clear warnings or instructions to farmers, contravening the law, according to experts.

Source: Taken from http://www.irinnews.org/Report.aspx?ReportId=87773 shortened by authors. Original article published by IRIN, the humanitarian news and analysis service of the UN Office for the Coordination of Humanitarian Affairs. [This article does not necessarily reflect the views of the United Nations]

must often accept working conditions that are unacceptable.

Direct causes of pesticide poisonings can be classified into two general categories that include several (functional) sub-categories which may relate to each other:

1. Unintentional poisoning

- Large scale accidents
- Improper use/storage
- Bystander exposure
- 'Proper' use
- Environmental contamination
- Residues in food

2. Intentional poisonings

- Suicide
- Homicide
- Intentional misuse

Pesticide residues in food can cause acute health effects (e.g. BfR 2011), but since Paraquat is not directly sprayed onto fruits and vegetables, and it does not move within plants, residues exceeding toxicological thresholds are not expected. The residues in raw produce following dessication are rather small (JMPR 1972, 1982). Therefore food residues are not a subject of this report.

Quite commonly, homicides as well as intentional misuse (abuse) using pesticides including paraquat (e.g. Stephens & Moormeister (1998); Daisley & Simmons (1999) and e.g. Beligaswatte et al. 2008, Garnier et al. 1994). For example, quite frequently people use, inappropriate pesticides for medical purposes (see paraquat cases with fatal outcome discussed in Garnier et al. [1994] and Binns [1976) . However, such intentional misuses are not subject of this report.

Suicides by ingesting/injecting pesticides and particularly paraquat are one of the most serious health issues in developing countries and are addressed in the Chapter 8.

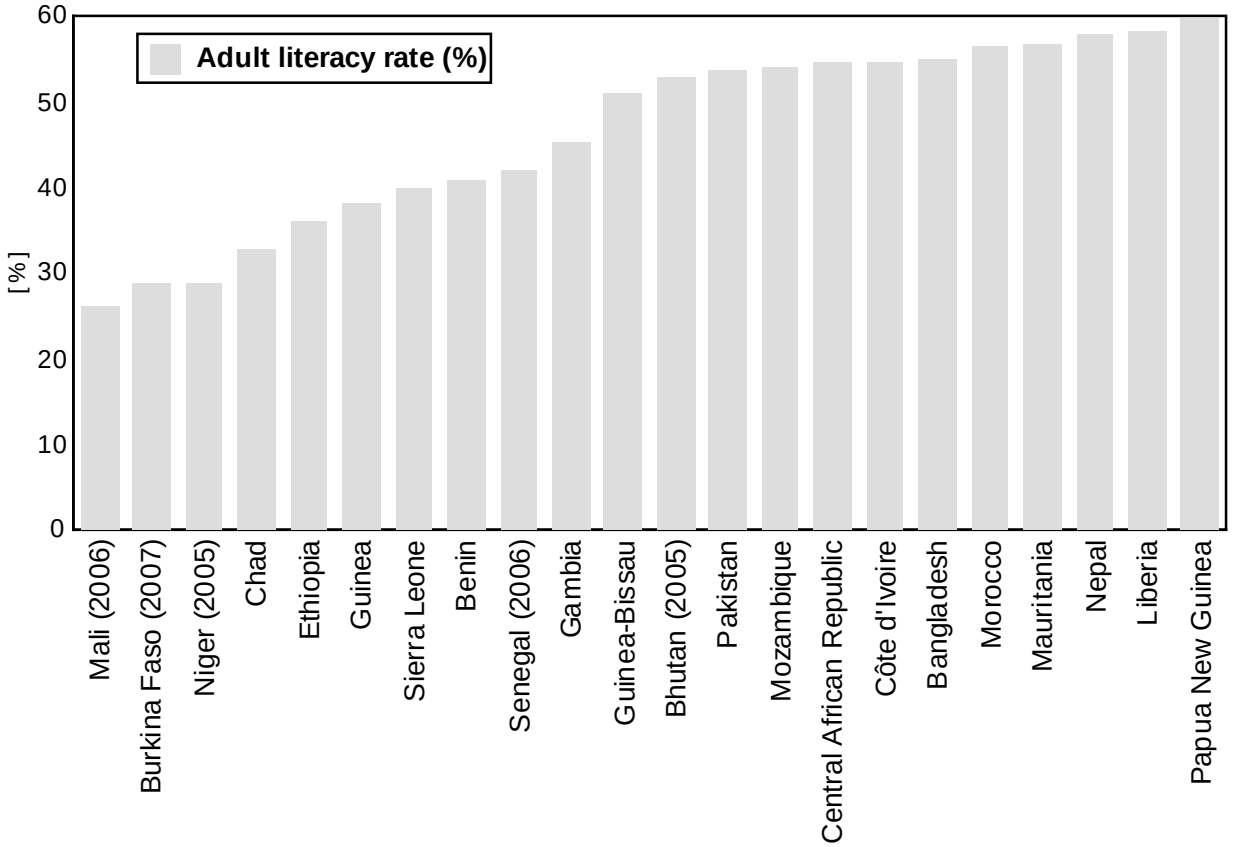

Figure 1: Countries with adult literacy rates below 60% in 2008 (deviating years in brackets) (UNESCO Institute for Statistic - Data Center - Predefined tables: http://stats.uis.unesco.org/unesco/TableViewer/tableView.aspx?ReportId=210

4. 1 Large scale accidents

There are different type of possibilities for accidents during the life cycle of a pesticide. The Bhopal catastrophe in India in 1984 was caused by the leakage of about 40 tons of methyl isocyanate (MIC) from a pesticide production facility causing approximately 15.000 fatalities and 100.000s of chronically ill people (Broughton 2004). Pesticide poisoning can also occur when during transportation of pesticides vehicles are involved in accidents (e.g. Cone et al. 1994).

However, most unintentional poisoning incidents are a consequence of improper use and storage.

4. 2 Improper use and storage

The term 'improper use' comprises numerous actions which violate 'Good Plant Protection Practice', which also includes good decision making for the selection of suitable pest control methods and preventive measures, e.g. within IPM.

4. 2. 1 Prior to application

Farmers in developing countries are often not able to make optimum decisions in pest management. The recognition of pests and their predators is generally rather low which leads to decisions to spray and kill any insect. Knowledge about product selection, optimized application rates and appropriate timing is often quite poor. Without sufficient knowledge of alternatives, farmers will often assume that the only solution to pest and weed problems is to spray more frequently (Dinham 2003, Williamson et al. 2008). 'Preventive' spraying on a regular/routine basis (e.g. by calendar) is still common in many places (Ngowi 2007).

Unsafe handling already starts with the purchase of pesticides that are not registered, illegal or outdated. This is common all over the world (e.g. see Balme et al. 2010). The business with illegal and counterfeit pesticides is so large even in Europe, that the European Crop Protection Association (ECPA) maintains a reporting web site[4].

4 http://www.illegalpesticides.eu/about/

Storage and Decanting

In February 2006, a family of three visited a relative's house in Morvell Australia. The two year-old son ingested the herbicide 'Spray Seed', which contains paraquat, from an unlabeled sports drink bottle.

The boy was initially treated for ingestion of the less potent herbicide 'Roundup' (containing glyphosate) and it was several days before doctors realised they were dealing with paraquat poisoning. By then it was too late for the little boy, who died six weeks later.[5]

Decanting pesticides into empty drinking bottles or food containers and/or using empty pesticide containers for food and drinks is still common (mal)practice in many countries and can cause severe poisoning (e.g. Leverton et al. 2007). Where pesticides are stored together with food confusion of bottles or other containers may occur.

Leverton et al. (2007) described cases in England in which pesticides were accidentally ingested because of poor storage:

- Weedkiller stored in a drink bottle
- Unlabeled bottle - thought to be lemonade
- Mistaken for painkiller
- Stored in a bottle thought to contain orange juice
- Mothball mistaken for a peppermint
- Wine placed in a can that had been used for weedkiller
- Mistaken for sweets
- Weedkiller thought to be fruit juice
- Mothball mistaken for a mint
- Packet unmarked
- Rodenticide and analgesics kept in same box
- Pesticide in an inappropriate container
- Insect repellent mistaken for sublingual medicine.

Considering the high toxicity of paraquat (see Chapter 5) and the absence of a effective medical treatment, such accidents involving

5 Steven G (2008): Poisoned. Article in the Latrobe Valley Express http://www.latrobevalleyexpress.com.au/news/local/news/general/poisoned/327709.aspx?storypage=1

Paraquat often are fatal (see Wesseling et al. 1997; Cravey 1979; Fernando et al. 1990).

Storage of pesticides in reach of children is a major cause of poisoning incidents involving children (Balme et al. 2010; UNEP 2004a).

Safety instruction

Illegal and repacked pesticides may come without label instructions, and might therefore not be used properly. Surveys have shown that label instruction when included are often not read by the users and/or not understood (Waichman et al. 2007; Damalas et al. 2006). In countries with a low literacy rates among adults (see Figure 1), written instruction may be useless for large parts of the rural population. This is particularly true for large numbers of plantation workers who are often migrants and cannot read the local language.

Pictograms as proposed by FAO do not replace education. A survey involving 115 farm workers in South Africa, showed that 50% or more of the farm workers had misleading, incorrect and critically confused interpretations of the label pictograms (Rother 2008).

4. 2. 2 Application

Studies in Africa show that many farmers mix several pesticides although no specific instructions either from the labels or extension workers regarding these tank mixtures existed (Dinham 2003; Ngowi 2007).

Corriols and Aragón (2010) describe practices involving pesticide handling reported at the time of acute pesticide poisoning children of working in agriculture, these practices are similar to those of adults:

- carrying pesticides,
- mixing and preparing pesticides,
- spraying equipment in bad condition with backpack leakage,
- applying pesticides by hand,
- lack of use of personal protective equipment,
- applying fumigants in a closed environment,
- planting fumigated seeds,
- eating and/or smoking without washing hands after exposure.

Most farmers and plantation workers in developing countries use backpack/knapsack sprayers. These backpack sprayers are frequently leaking - only 48% of over 8.500 smallholders interviewed in 26 countries

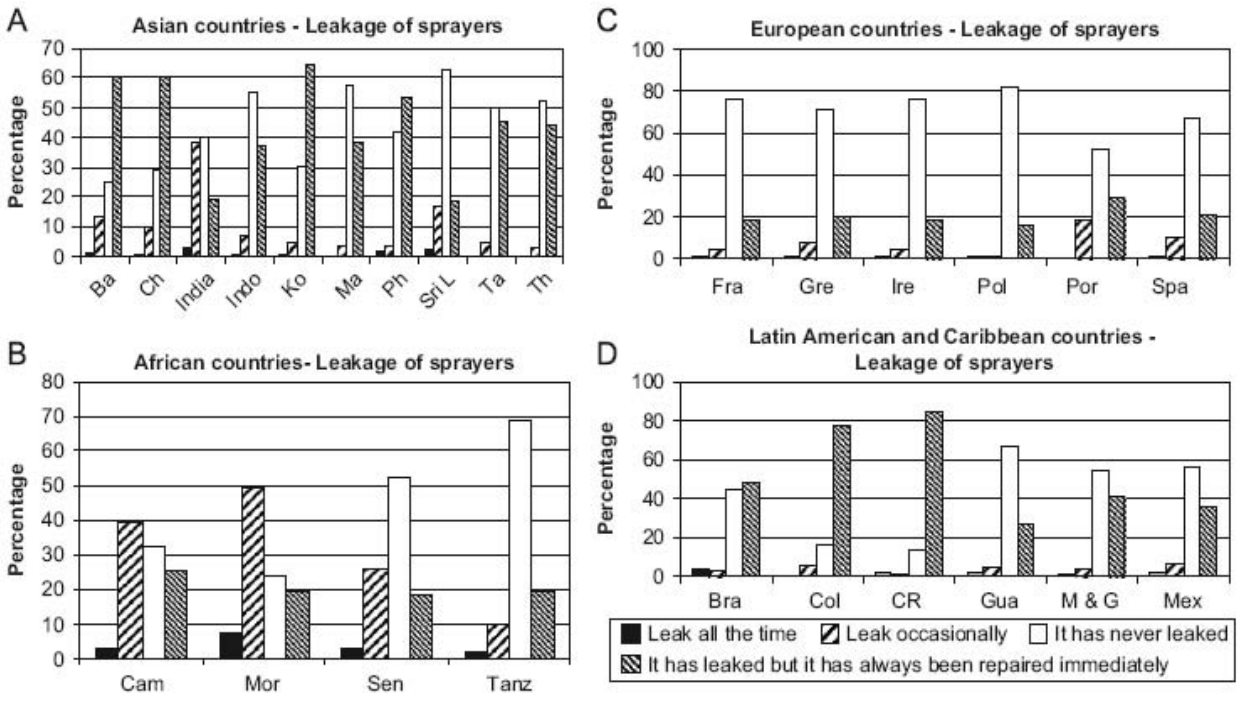

Figure 2: Leakage of sprayers in (A) Asian, (B) African, (C) European and (D) Latin American countries. (Matthews 2008).

reported that their sprayers had never leaked. A further 38% reported they were able to repair any leaks immediately. In Morocco, Cameroon, Senegal and India, 38% of users reported that occasional leaks were not always repaired immediately (see Figure 2). The reasons quoted were lack of importance attached to the need to avoid leakage of sprayers plus lack of spare parts, knowledge and cost (Matthews 2008).

The same study also showed that a very high proportion of interviewed farmers in Asia, especially in Bangladesh, India, Philippines and Sri Lanka, do not wear the minimum protective clothing consisting of long-sleeved shirts and long trousers and shoes or boots while spraying (see Figure 3).

Only 20% of all respondents (in developed as well as in developing countries) wore the recommended five key items, including long trousers, and long sleeved shirts (or overalls), gloves, boots and face shield while mixing and loading pesticides (ibid.). In most cases, because overalls are an extra expense, some form of normal clothing is used.

Another survey, in Greece showed that, although 99% (or 223 in total) of the surveyed farmers thought that pesticides can have serious adverse effects on users' health, 46% reported not using any special protective equipment when spraying pesticides. Only few farmers reported using a face mask (3%), gloves (8%), and coveralls (7%) on a regular basis (Damalas et al. 2006).

The reasons for not using protective equipment during pesticide handling were that protective equipment is uncomfortable, too expensive to buy, time-consuming to use, not available when needed, and not necessary for each case (ibid.). These are reasons commonly mentioned by user across the world.

A small survey by NGOs in Pakistan, Indonesia and China showed that many pesticide dealers selling paraquat do not sell protective gear and cannot tell customers where to they might find such items. In Pakistan, none of the paraquat dealers sold PPE, nor did they know, where to purchase them, the situation was only slightly better in China. In Indonesia, the surveyed stores were comparatively well equipped (Dinham 2007).

A study conducted in the Philippines showed that certain beliefs prevented the use of PPE:

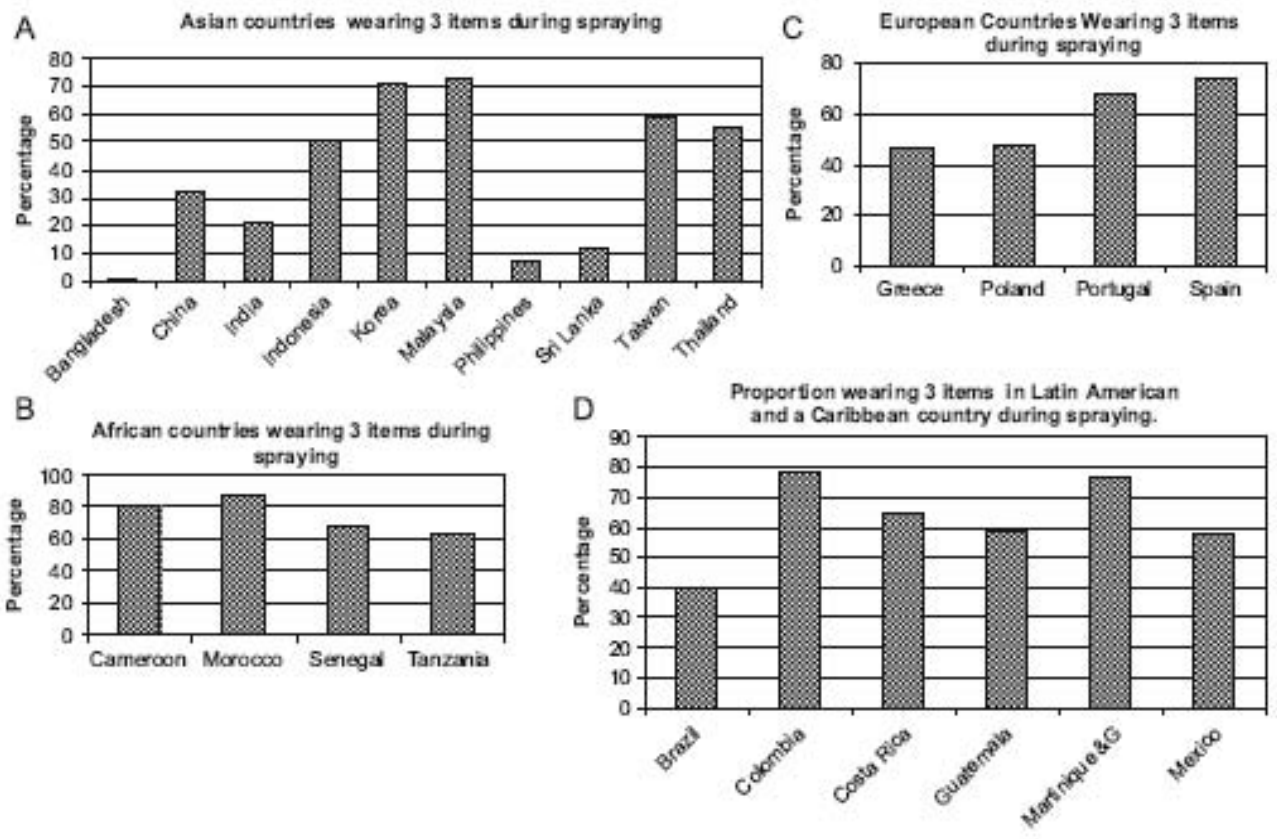

Figure 3: Proportion of users of three items of PPE during spraying in (A) Asian, (B) African, (C) European and (D) Latin American countries. (Matthews 2008).

Box 2: Occupational Pesticide Exposure

Pesticide exposure occurs both while workers prepare the pesticide for application (during mixing and loading) and while they actually apply the pesticide. Equipment cleanup and repair may also contribute to exposure. These separate tasks may be done by different people or a single individual may combine them. Different formulations, handling methods (closed-system design), application methods (high pressure spray vs. granular) contact to the sprayed plant and temperature etc. may affect levels of exposure (Rutz & Krieger 1992; Machera et al. 2003). In general, more manual handling and less protective measures increases exposure. Hand spraying, for example proved to present the greatest risk of exposure of the methods of application studied.

The main route of pesticide exposure for agricultural workers is through the skin. During mixing and spraying of pesticides, 87–95% of overall exposure was seen to arise via the skin, while inhalation accounted for 5–13% of exposure, and manual sprayers clearly caused the greatest exposure with a mean rate of 1.040 mg/h (Rutz & Krieger 1992).

A trial with a low pressure knapsack sprayer with two operators showed a body contamination of 25.37 and 35.83 ml/h plus a hand contamination of 74.27ml/h and 115.02 ml/h spray solution, whereas the leading hand was significantly more exposed. In the case of hand lance applications with tractor–generated high pressure (n=3) the body contamination ranged from 160.76 to 283.45 ml/h spray solution, while the hand contamination ranged from 3.71 to 4.53 ml/h. The high pressure application lead to higher inhalative exposure 0.34-0.62ml/h compared to 0.067–0.086ml/h for the low pressure application (Machera et al. 2003).

One study looking are tractor mounted spraying ranged daily potential dermal exposure from 2.0 to 567.8 mg active substance (median = 57.8 mg) in 47 farmers. Exposure during mixing–loading tasks accounted for 13.9–98.1% of the total exposure (median = 74.8%). For mixing–loading, hands and forearms were the most contaminated body areas accounting for an average of 64 and 14%, respectively. For application, hands were also the most contaminated part of the body, accounting for an average of 57%, and thighs, forearms and chest or back were in the same range as one another, 3–10% (Lebailly et al. 2009).

The percentage of **paraquat** absorbed through intact human skin (arm, leg or hand) is estimated to be 0.23-0.29% (Wester et al. 1984), although poisoning cases through intact skin are reported (Peiró et al. 2007, Tungsanga et al. 1983). Skin is more vulnerable when it has been injured or is damaged, for example through contact with paraquat. Absorption via the skin is also higher in workers who have dermatosis (Garnier 1995). In certain areas of the body, skin is highly permeable, e.g. in the genital area exposure can result in a 50 times greater absorption (Semple 2004). It was found that sweat on skin from perspiration led to increased skin absorption (Williams et al. 2004). Several paraquat poisoning cases via dermal exposure have been reported (see Chapter 6 and 7).

The Scientific Committee on Plants (SCP) of the European Commission (EC) commented on the risk to workers taking into particular account potential inhalation and skin exposure. Estimates based on exposure models suggested that exposure of knapsack sprayers to paraquat may exceed the shortterm Acceptable Operator Exposure Level (0.0005 mg/kg b.w./day) 60 times with protective (AOEL) equipment and 100 times without it (EC 2002a). It was concluded that even with gloves, breathing equipment, overalls, wide-brimmed hats and solid shoes, the level of exposure is above the AOEL (EU 2007).

Figure 4: Unprotected paraquat user in China. The plastic cover on the back should prevent exposure from a leaking knapsack, but in the same time, no shoes and gloves are worn.
© PEAC China

non-users of PPE responded that they were immune or not susceptible to pesticides because ‚their blood is strong or their blood can take pesticides'. In addition, most of the respondents believed that pesticides might only be dangerous through inhalation and oral ingestion (Palis et al. 2006).

4. 2. 3 Post-application

Washing

If agricultural workers do not wash their hands after applying pesticides exposure might continue, esp ecially when no gloves were worn. Corriols and Aragón (2010) report poisoning incidents due to workers eating without having washed their hands after exposure. Matthews (2008) reports that the majority of over 8.500 respondents said they had a shower or washed immediately after spraying (77%), with a further 10% 'often' taking showers after an application. This high rate may relate to the hot climate in many countries, which more or less necessitates washing or taking a shower after work in the field anyway. In colder region this percentage might be lower (e.g. Ireland see Matthews 2008). A survey in Nepal showed that a total of 52% females and 42% males have not had shower after sprays (Kishor 2007).

Disposal

Empty pesticide containers might be re-used, thrown into the field, burnt or buried (Ntow et al. 2006). Usage of empty containers for food and beverages is a major cause of accidents (see previous Chapter on Storage and Decanting). Empty pesticide container not only pose a threat to the environment, but also to people, for example children who may use them to play.

4. 3 Bystander exposure

Bystanders are persons who are not directly involved in a specific pesticide application. Bystander exposure might be a consequence of improper use such as no warning signs on freshly sprayed fields, aerial over-spraying of villages, violation of re-entry periods. But bystanders can also be exposed during 'proper' use of pesticides (e.g. through spray drift). Affected bystanders can include residents of communities living near fields or in plantations, unprotected workers working simultanously in field while a spraying operation is being conducted, and people walking through a recently sprayed field. Walking through a sprayed field can lead to significant exposure especially to the legs (Snelder et al. 2008, Farahat et al. 2010). Entering a recently sprayed site can lead to acute poisoning due to the evaporation of pesticides (Corriols & Aragón 2010).

From 1989 to 1992 in the UK, for example, 129 cases of non–fatal pesticide poisoning were rated as 'confirmed' or 'likely'; 41% of confirmed cases were people living beside a sprayed field; 35% were working with a pesticide or standing close to a user and 23% passed by fields that had recently been sprayed (Thompson et al. 1995b).

4. 4 'Proper' use

Acute poisoning should not occur, if pesticides are used according to the instructions, but exposure cannot always be avoided. Personal protective clothing is permeable (e.g. see Protano et al. 2009; Machera et al. 2009; Machera et al. 2003; Vitali et al. 2009). In the daily routine small spills and splashes happen even when experienced users apply pesticides. Applicators may also inhale pesticides, if a breathing mask is not required.

Penetration of clothing by various pesticides including paraquat was tested for different types of fabric. It was found that shirting or lightweight fabrics provided the least protection, while heavier weight fabrics (denim and twill) offered significantly greater protection. Normal work clothing did not give sufficient protection from heavier spray or a spills (Branson & Sweeney 1991). It was found that shirts (cotton/polyester) became wet and clung to the skin, which resulted in significantly greater exposure than with double-layer cotton coveralls. Considerable exposure also occurred through openings at the neck and sleeves (Fenske 1988).

Protective clothing also has limited effectiveness, when spills or knapsack leakages occur soaked cotton will prolong skin exposure. In the court case about the authorization of paraquat in the EU *'(...) the Republic of Finland points to a case of an operator whose trousers were stained with*

paraquat while transferring the substance from one receptacle[6] to another and who waited 48 hours before cleaning the stain in question. Ten days after the incident, his lungs ceased to function and he died on the 15th day after the incident (...)' (EU 2007 pg. II - 2463).

Basically each pesticide user should have either non-permeable protective clothing and/ or have a second set of protective clothing to change.

Lee et al. (2009) detected urinary paraquat during spray days, although most plantation workers wore protective clothing (see Chapter 6.1 item o)), and the Court of First Instance of the European Communities (EU 2007) concluded that even when protective clothing is worn, paraquat exposure can exceed the acceptable levels for users (see Box 2).

6 Container (Footnote by the authors)

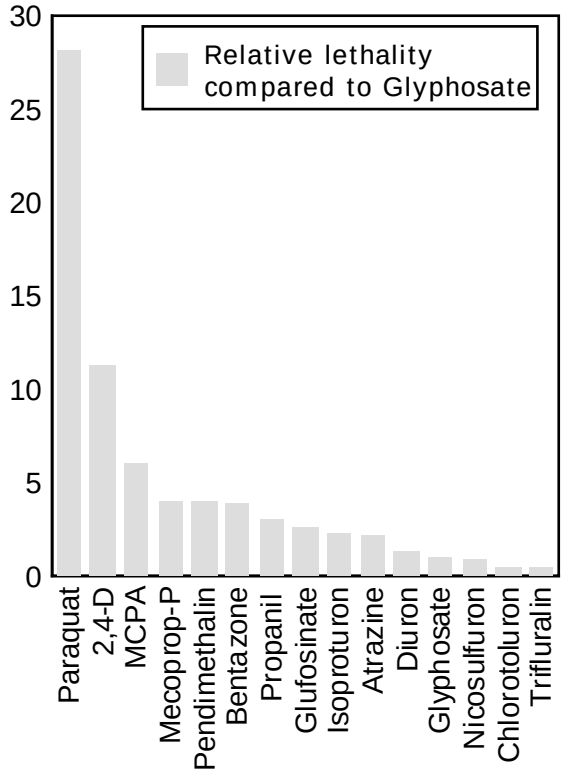

Figure 5: Relative lethality of 15 common herbicides based on LD_{50} by WHO 2010 to Glyphosate (LD_{50} of 4,230 mg/kg*b.w. = 1). LD_{50} values indicated with >5,000 or >10,000 were calculated with 5,001 and 10,001 respectively. Own graphic based on WHO (2010)

5. Paraquat – the deadliest pesticide

Fatal incidents involving paraquat occurred from the time when it was first used. In 1979, Fischer and Kahler cited 232 fatal cases globally, of which 109 were suicides, 96 accidents, and 27 poisonings with an unknown case history (Fischer & Kahler 1979). These figures appear to be low compared to the estimated 2,000 suicides committed annually with paraquat in South Korea alone (Seok et al. 2009), but it needs to be considered that reporting of poisonings was much less developed in the 1960ies and 70ies than today. Dawson and Buckley (2007) speak of ‚*tens of thousands of deaths worldwide*' due to paraquat poisoning each year.

There are three things which make paraquat the deadliest pesticide when looking at acute pesticide poisoning statistics:

1. The high acute toxicity to humans – paraquat is about 28 times more acutely toxic (based on oral LD_{50}) than the globally most widely used pesticide glyphosate, and much more acutely toxic than all common herbicides (see Figure 5). In the court case Sweden vs. the European Commission, Sweden '*(...) claims that paraquat is the substance most dangerous to health — in terms of acute toxicity — ever included in Annex I[7] to Directive 91/414 (...)*' (EU 2007 pg. II–2462). Figure 6 shows the mortality rate for pesticides used for deliberate self–harm in Sri Lanka and other countries.[8] Paraquat has the highest mortality rate, over 40% of the people ingesting it, die. In Japan, the mortality rate is even over 70% (Nagami et al. 2007).

2. The absence of an antidote against paraquat poisoning.

3. Its potential for being absorbed through skin after prolonged exposure – if skin has been damaged.

7 Annex I is the positive list of pesticide active ingredients of the European Union (EU). Only pesticides products containing active ingredients of Annex I can be authorized in the EU. The inclusion of paraquat to Annex I was annulled by the court and paraquat use is banned by now. (Footnote by the authors.)

8 Since Endosulfan and WHO Class I pesticides (e.g. Monocrotophos, Methamidophos) are already prohibited in Sri Lanke, mortality rates were taken from and India resp. Brazil.

The widespread availability of paraquat is one important reason for the massive numbers of fatalities, but other, more common pesticides cause much less fatalities (see Figure 6).

Paraquat is classified in WHO class II for acute hazard based on an oral LD_{50} in rats of 150 mg per kg body weight (b.w.) (WHO 2010), but an oral ingestion of a mouthful of the 20% solution is likely to cause death, and depending on the constitution of a person 10 ml are enough to be fatal (Bismuth et al. 1982).

An estimate of a minimum lethal human dose for paraquat dichloride is approximately 46 mg/kg b.w. (equivalent to 33 mg cation/kg b.w.) (Pasi 1978). Individuals vary in sensitivity and tolerate different doses. Minimum fatal doses by ingestion of concentrates (12–20%) are 30–50 mg/kg b.w. for paraquat dichloride, corresponding to a single swallow (Bismuth et al. 1995). This range is also confirmed by KEMI 2006. Table 1 shows different LD_{50} values compiled by KEMI (2006).

Humans are more sensitive than rats. The intake of 17 mg cation/kg b.w. (equivalent to 23.5 mg/kg b.w. of paraquat dichloride) has been fatal (Stevens & Sumner 1991). After ingestion of more than 15 ml (one tablespoon) of 20% concentrate, the outcome is most likely to be fatal (Pronczuk de Garbino 1995). While the body can dispose of lower doses, a large dose (20 mg/kg b.w.) damages the kidneys, reducing the possibility of disposal (Houze et al. 1995).

Table 1: Oral LD50 of Paraquat for different mammalian species (KEMI 2006)

Mammal species	Oral LD_{50} (mg/kg*body weight)
Rat	40–200
Guinea pig	22–80
Rabbit	49–150
Sheep	50–75
Cat	26–50
Dog	25–50
Monkey	50
Human	40–60

Numerous scientists dealing with paraquat poisonings argue that '*it would be appropriate to assign paraquat to [WHO][9] Class I, like captafol, which was categorized as Class Ia by reason of carcinogenicity.*' (Nagami et al. 2005 pg. 183, see also Nagami et al. 2007 and Dawson et al. 2010).

There are more flaws in the WHO classification system for the acute toxicity of pesticides. Paraquat (dichloride) has been classified 'Fatal if inhaled' (Hazard Classification 330)[10] and 'Very toxic by inhalation' (Risk Phrase 26) by the European Union (EU 2008), but the WHO does not consider inhalative exposure at all.

6. Occupational exposure

Exposure to paraquat depends on application technique, protective measures, spray solution, frequency of use as well as on environmental conditions (see Box 2).

It has been estimated that workers on large plantations spray herbicides such as paraquat during more than 1,400 hours per year (Whitaker 1989). This means that workers spray for over 175 working days a year. Women in Malaysian plantations spray on average 262 days a year. Similar numbers have been given by Matthews (2008). He estimated that Indonesia contractors were undertaking between 640 and 1,380 hours of spraying (all pesticides) per year using knapsack sprayers. In Malaysia, the female tree plantation workers targeted sprayed for up to 1,540 hours per year. This is equivalent to spraying for 6.5 hours per day for 240 days per year with a knapsack sprayer (ibid.). While these numbers do not refer to paraquat use only, a majority of spraying hours might be with paraquat, because weed control in tree plantations is the most dominant application of pesticides, and paraquat is still one of the preferred chemicals for weed control in tree plantations especially in palm oil.

In field studies, the US Environmental Protection Agency found that margins of exposure to paraquat for workers using low pressure sprayers or backpack sprayers were unacceptable and that the practicality of additional personal protective equipment required to reduce health risks was a matter of concern (US EPA 1997) (study d) below).

9 Addition by the authors.

10 Based on an LC_{50} inhalation for mist/dust of 0.05–05 mg/litre/4h.

In a study with workers applying paraquat with knapsack sprayers, the absorbed doses based on dermal exposure were 0.0004-0.009 mg/kg bw–day, which is up to 18 times higher than the proposed short-term Acceptable Operator Exposure Level (AOEL) of 0.0005 mg/kg bw–day. The absorbed doses that were estimated from urine and blood analyses were 2 to 8 times above the AOEL (EC 2002 and reference therein: Chester et al. 1993, study e) below). In another study the mean absorbed dose was 0.00015 mg/kg bw–day or 30% of the AOEL (Findley et al. 1998).

Within the EU review of paraquat, the Scientific Committee on Plants (SCP) commented on the risk to workers taking into particular account potential inhalation and skin exposure. Estimates based on exposure models suggested that exposure of knapsack sprayers to paraquat may exceed the short-term Acceptable Operator Level (0.0005 mg/kg bw–day) 60 times with protective equipment and 100 times without it (EC 2002a).

Monitoring workers' exposure in the field indicated that exposure estimated in the models was higher than the actual exposure. Also that workers absorbed high doses when they did not use the recommended protection (gloves and other protective clothing) (EC 2002a).

6. 1 Evidence of exposure

a) Machado–Neto et al. 1998

Studies on the efficacy of safety measures for knapsack sprayers applying paraquat to maize were carried out. It was found that spraying in front of the workers' body was not safe. The potential skin exposure with spray was too high: 1,979.8 and 1,290.4 ml/day for a 0.5 m long lance (shaft) and for a 1.0 m lance, respectively. Based on calculated margins of safety[11], it was estimated that potential skin exposure needed to be reduced by 50–80% for a 0.5 m lance, and by 37–69% for a 1.0 m lance.

11 Margin of safety: ratio of the highest estimated (or actual) level of exposure to a pesticide and the toxic threshold level (usually the no–observed effect level) (Holland 1996)

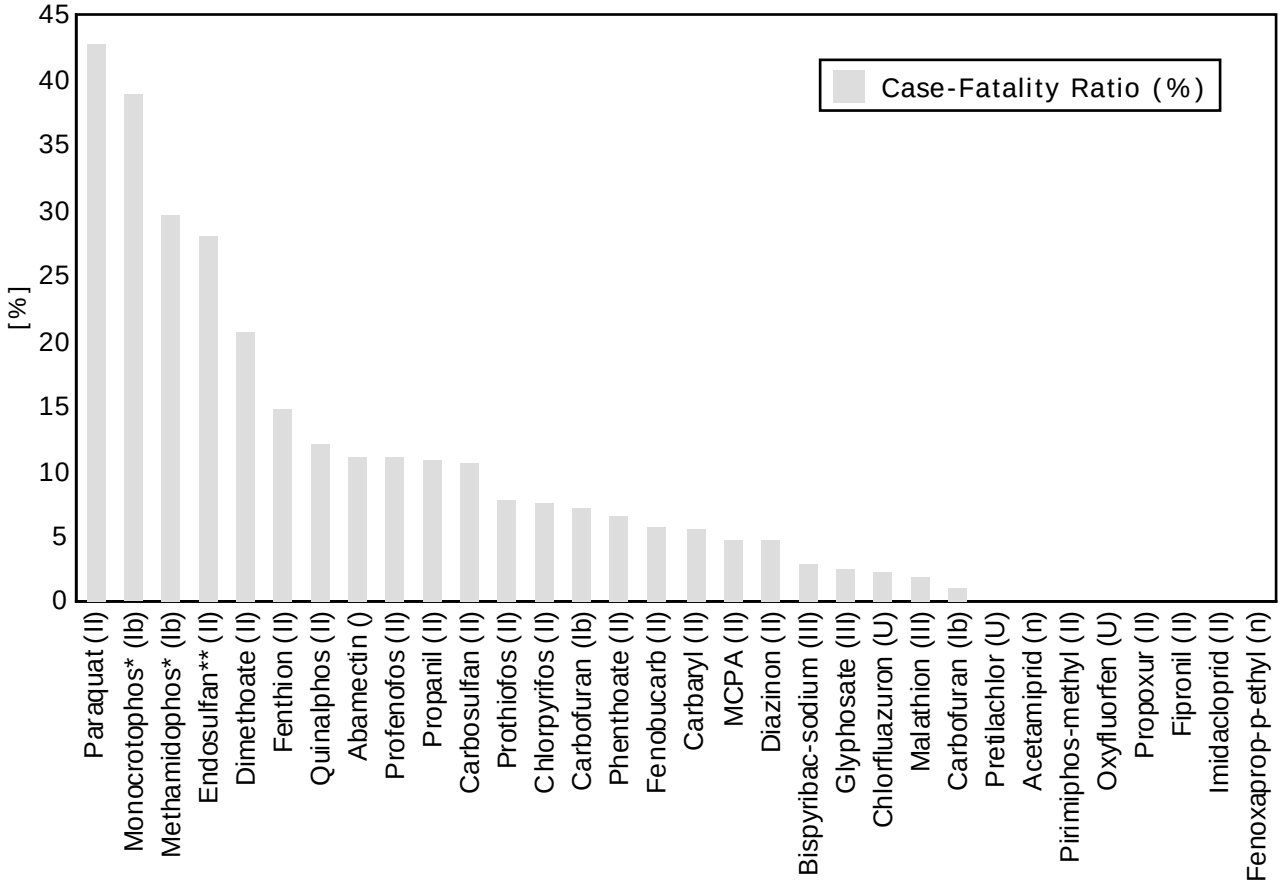

Figure 6: Case-Fatality Ratio (%) for deliberate poisonings with pesticides in Sri Lanka (Dawson et al. 2010), *Brazil (Recena et al. 2006) **India (Srinivas Rao et al. 2005). WHO classification: Ib= Highly hazardous;II = Moderately hazardous; III = Slightly hazardous; U = Unlikely to present hazard in normal use. Pesticide without WHO classification = n. Only pesticides > 10 cases are presented.

Potential skin exposure was significantly reduced when the spray lance was placed behind the worker (attached to the backpack) as most of the potential exposure arose from sprayed plants contaminating the skin of legs and feet. A longer spray lance alone did not reduce the potential skin exposure enough to provide safe conditions. Workers who mixed solutions and loaded them into tanks received the main exposure at the hands. Although mixing and loading was considered to be safe, it was recommended that impermeable gloves should be used as a further safety measure.

b) van Wendel de Joode et al. 1996

A study on banana plantations in Costa Rica measured the exposures of 11 spray applicators to diluted paraquat (0.1–0.2%). Total skin exposures (sum of certain body areas) were 0.2–5.7 mg paraquat per hour (equivalent to doses of 3.5–113.0 mg/kg). Urinary levels (detected in 2 of 28 samples) were <0.03 mg/l and 0.24 mg/l. Respiratory exposure was 0–0.043 mg/l, corresponding to 0.3% of total dermal exposure.

It was found that the risk of high and therefore hazardous exposure was continually present, due to poor working conditions. Health problems recorded were:

- blistering and burns on hands, thighs, back, testicles and legs (due to defective equipment or contact with sprayed leaves);
- two eye splashes causing redness and burning sensation;
- three workers had nosebleeds (in one case frequently);

c) Spruit & van Puijvelde 1998

A study in Nicaragua found lower paraquat levels than in study b (above), but residues on skin were still considerable, especially on the hands. Workers did not use adequate protection.

d) US EPA 1997a, pg. 56

In a study in the US on the exposure of workers who mixed, loaded and applied paraquat, it was concluded that the margins of skin exposure (the No Observed Effect Level [NOEL] divided by total daily dose) were unacceptable for backpack applicators and workers who used low pressure sprayers even when they wore long pants, a longsleeved shirt, chemical resistant gloves and shoes with socks as personal protective equipment (PPE).

This type of PPE is required for applicators and other handlers. Additional PPE–a chemical resistant apron and face shield–is required as minimum standard by the Environmental Protection Agency for mixers and loaders who handle paraquat products. EPA stated that it was 'concerned about the practicality of adding another layer of PPE (woven material), due primarily to heat stress considerations'.

e) Chester et al. 1993

In Sri Lanka a study with 12 workers who applied paraquat at a concentration of 0.03-0.04% (cation wt/vol) with knapsack sprayers measured skin exposure and urinary levels. The mean potential skin exposure for workers who mixed and loaded spray solutions was 66 mg (per day). For spray operators it was 74 mg. The workers did not wear protective clothing.

The proportion of the total potential exposure deposited on skin was estimated to be about 95% for mixer/loaders (86% on the hands) and about 90% for spray operators (on the hands, legs and feet). Urinary paraquat was mostly below 0.1 µg/ml, with a maximum of 0.37 µg/ml. The extent of absorption was low due to the very dilute spray solution and high standard of personal hygiene.

f) Seiber et al. 1983

In this study it was found that paraquat residues on cotton plants 4 weeks after application gave rise to concentrations in air of up to 0.47–1.2 pg/m3 close to the harvesting tractor. These resulted in an estimated maximum exposure by inhalation of 16.3 pg per day (based on an average breathing rate of 1.7 m3/h for light work and an 8h–working day). The upper value of paraquat air concentrations would result in an exposure corresponding to 43.5% of the Acceptable Operator Exposure Level (AOEL) for a worker weighing 75 kg. 70% of the airborne paraquat in dust had respirable size. Skin exposure was not measured. Substantial skin contact with the dust could have considerable impacts on the overall exposure. The residues of paraquat in air surrounding a harvesting tractor were sufficiently high to argue for the required use of closed cabin harvesting tractors.

g) Howard 1982; Howard et al. 1981

A study with 14 workers in Thailand who used knapsack sprayers or low–volume spinning disc applicators (with spray concentration 0.15% and 0.2%), measured urinary paraquat of 0.73–10.21 mg/l after 14 days spraying. Levels were significantly higher in unprotected men. And levels in urine increased as the trial progressed. Irritation of unprotected skin was severe (caustic burns to the feet) in workers who used low–volume applicators (higher concentration).

In a study in Malaysia of 27 workers who had sprayed paraquat (0.1% cation content) for at least 1000 hours, 11 reported one or more incidents of rashes or skin irritation that were associated with spraying, mostly on the hand, legs or in the groin, and there was one case of eye injury.

The transfer factor (diffusion in the lung) was 4.9–7.3% lower among the sprayers than among non–exposed or general factory workers, (although not statistically significant).

h) Chester & Woollen 1982

A study in Malaysia detected urinary paraquat in 9 out of 19 workers spraying paraquat (0.1–0.2% solution content of the cation) and in 1 out of 7 mixers (who mixed the solution). Urinary levels were below or equal to 0.05 mg/l in 12 of the 19 spray operators but ranged up to 0.69 and 0.76 mg/l. The contamination of the body was highest on the hands.

Paraquat was detected in a small proportion of workers who did not handle paraquat but entered sprayed areas. The average exposure for uncovered skin was an estimated 2.2 mg per hour (ranging from zero to 12.6 mg/h). For unprotected skin and clothing combined it was 66.0 mg/h (range 12.1–169.8 mg/h); the proportion of paraquat from clothing that reached the skin was estimated as 5%. The mean (average) skin exposure was 1.1 mg/kg bw per hour, and the highest individual total exposure was 2.8 mg/kg b.w. per hour. In air, the mean paraquat concentration was 0.24–0.97 pg/m3 (equivalent to 1% or less of the threshold limit value, 0.1 mg/m^3).

i) Kawai & Yoshida 1981

Workers who were exposed to concentrations of paraquat in air of 0.011–0.033 mg/m^3, and who had worn gauze masks, had 1.4–2.7 pg/l paraquat in urine 24 hours later. But none was detected in workers who had worn a high–performance mask. The spray concentration was 0.08% paraquat (24% solution diluted 300 times); total skin exposure was about 0.22 mg. The need for protective equipment

Box 3: Acceptable levels of exposure – ADI, RfD & AOEL

An acceptable daily intake (ADI) denotes '*an estimate of the daily exposure dose that is likely to be without deleterious effect even if continued exposure occurs over a lifetime*'. The toxicity reference dose (RfD) is another term for this (WHO 2004a). For paraquat the ADI is 0.006 mg per kg body weight (bw) per day for the dichloride salt, or 0.005 mg/kg bw–day for the cation (FAO 2004a). The reference dose established in the US is 0.0045 mg cation/kg bw day (US EPA 1991). The European Commission established an ADI of 0.004 mg/kg bw–day (EC 2003b).

An ADI or RfD represents a 'very low risk' intake, or dose, but it is not possible to define what 'very low' means. For susceptible individuals a harmful effect may appear at lower doses than the ADI (Rodricks 1992). For children the Californian EPA proposed a RfD of 0.00007 mg/kg bw–day, which is based upon a neurological study (Cal EPA 2009), which is by magnitudes lower than the RfD for adults.

The Acceptable Operator Exposure Level is according to the European Union (EU 1997): *'(...) the maximum amount of active substance to which the operator may be exposed without any adverse health effects. The AOEL is expressed as milligrams of the chemical per kilogram body weight of the operator. The AOEL is based on the highest level at which no adverse effect is observed in tests in the most sensitive relevant animal species or, if appropriate data are available, in humans (...)'*. The AOEL can be distinguished for several exposure paths (inhalation, skin, oral). If all exposure paths are considered a 'systemic' AOEL is applied.

For paraquat the systemic AOEL is 0.0005 mg/kg bw–day for shortterm exposure and 0.0004 mg/kg bw–day for longterm exposure (EC 2003b).

to reduce skin and inhalation exposure was highlighted.

j) Swan 1969

Paraquat was detected in 24.8% of the urine samples of 30 workers in two studies in Malaysia. The workers sprayed a 0.05% paraquat solution over a 12 week period. Peak (mean) levels measured were 0.32 (0.04) mg/l and 0.15 (0.006) mg/l, respectively.

k) Hayes & Laws 1991

Skin exposure to paraquat measured during proper application with either pressurised hand sprayers or tractor–mounted sprayers (low boom) ranged up to 3.4 mg/h. Practically all of the skin contamination was found on the hands. Inhalation exposure ranged up to 0.002 mg/h.

l) Baselt 1988, and Baselt & Cravey 1989

Studies in the US with workers who applied paraquat (0.25%) over a 12 week period found urinary levels of paraquat between 0–0.15 and 0.32 mg/l (average was below 0.04 mg/l).

m) Staiff et al. 1975

In the US an average exposure of 0.40 mg/h (range 0.01–3.40 mg/h) was measured for workers using a tractor–mounted sprayer, and 0.29 mg/h (0.01–0.57 mg/h) for the use of pressurised hand dispensers. Solutions contained 1.2% and 0.2% paraquat, respectively. No detectable levels of paraquat were found in urine (limit of detection was 0.02 mg/l). With both ways of application practically all of the skin contamination was found on the hands. The average exposure by inhalation was below 1 pg/h (range 0–2 pg/h and 0<1 pg/h).

n) Wojeck et al. 1983

In a study with workers using tractor–drawn sprayers (with a drop boom) average exposure to uncovered skin and clothing combined was 168.59 mg/h in tomato fields (spray solution 0.05% paraquat); average inhalation exposure was 0.07 mg/h. Exposure was lower with an enclosed or high–clearance tractor. In citrus groves, average exposures to skin and clothing were 12.16 mg/h (spray 0.05%) and 28.5 mg/h (spray 0.11%). Workers wore a shirt, long pants, socks and shoes/boots. A level of 0.033 mg/l paraquat was measured in one urine sample.

o) Lee et al. 2009

Box 4: Protective Equipment needed to use Paraquat

Applicators and other handlers using 'Gramoxone Inteon' (ca. 30% paraquat dichloride content) in the US must wear long–sleeved shirt, long pants, shoes plus socks, protective eyewear, chemical resistant gloves (Category A), plus adust mist NIOSH[1]-approved respirator with any N, R, P, or HE filter. In addition to that, mixer and loaders must wear a chemical resistant apron and a face shield (Syngenta 2010).

In Malaysia the label of one paraquat product gave the following directions: 'When using product, wear protective clothes including gloves, mask'. Eye protection and a respirator (breathing mask with filter) were not prescribed (Crop Protection 2004). On the labels of paraquat products sold in Thailand it was prescribed to wear footwear, mask and gloves while spraying and to wear mask and gloves during mixing and to wear boots, eyeglasses and gloves when holding or transporting the product.

The label of a paraquat product sold in Mexico requires use of industry glasses and a mask for dusts or mists, chemical resistant gloves, an overall, hat and rubber boots. It is recommended to apply the product (25% paraquat dichloride wt/vol) at a rate of 2.0 to 3.0 l/ha diluted 'in the sufficient quantity of water' (Syngenta 2004).

In Malaysia, Thailand and Mexico, maximum recommended concentrations were below or equal to the maximum recommended concentration in the US for backpack application. However, on all products the information was less comprehensive and the personal protective equipment required was generally less extensive than in the US.

1 The National Institute for Occupational Safety and Health (NIOSH)

Urine excretion of paraquat was measured in paraquat users (knapsack) and non-users working in coffee, banana and palmoil plantations in Costa Rica. Potential inhalative exposure was measure for a subset of the tested workers. Urinary paraquat measurements were non-detectable or very low when workers did not handle paraquat. Despite some protective clothing[12] 53,9% of the paraquat users excreted paraquat during the day of spraying, which prooves exposure. The average (±Standard Deviation [SD]) of urinary paraquat excretion level on days when workers handled paraquat was 6.3 (±10.45) µg/24 h (Coffee: 5.74 [±10.13]; Banana: 11.39 [±13.48]; Palmoil: 2.19 [±1.94]). Paraquat exposures among handlers on spray day were significantly associated with the type of crop, whereas length of spray day is probably the important contributing factor.

The average (±SD) for inhalable dust exposure was 218.86 (±253.50) µg/m3 air. For Airborne paraquat the average exposure was 6.07 (±4.77) µg/m3 air. These levels were significantly lower than the occupational exposure standard.

p) Morshed et al. 2010

In an experimental trial in Malaysia, Morshed et al. (2010) measured airborne paraquat before, during and after spraying. They measured the highest paraquat air concentration during the 25 min. spray application at operator's breathing zone, which was with 125 µg/m3 above the TLV (threshold limit value) and REL (recommended exposure limit) (100 µg/m3) of ACIGH (American Conference of Government Industrial Hygienists) and NIOSH (National Institute for Occupational Safety & Health). The acceptable operator exposure level (AOEL) of 0.0005 mg/kg bw–day was theoretically exceeded when extrapolating air residue data to potential dermal and inhalation exposure.

7. Acute health effects of paraquat

7. 1 Acute systemic poisoning

The exposure of farmers and agricultural workers to paraquat, during mixing and spraying, has acute (immediate) toxic effects and chronic (long-term) effects on health. Acute health effects occur frequently among paraquat users. They include eye injury, nosebleed, irritation and burns of skin. In case of acute paraquat poisoning, difficulty in breathing may develop with a delay of two to three days; death can occur up to several weeks after absorption.

Systemic poisoning denotes an incident of exposure to a toxic substance that is followed by symptoms due to absorption by the system and ensuing damage of organs. The term 'poisoning' includes incidents of exposure that lead to skin or eye damage, irritate the upper airway and cause nosebleed, and to exposures that result in the systemic absorption of the toxic agent referred to more specifically as '*acute (systemic) poisoning*'.

The toxic effects of a substance absorbed depend on specific modes of action in an organism (distribution, storage, metabolism, reversible or irreversible effects, excretion), physical state, the amount absorbed (depending on volume, concentration and duration of exposure) and individual susceptibility (body weight, health and other factors) (Frumkin 2000). The route of absorption has an indirect impact, as it influences the amount absorbed, besides causing irritant and harmful effects.

When paraquat is absorbed through skin it can lead to systemic poisoning with the same features as those resulting from ingestion. Prolonged contact with paraquat (from leaking equipment or soaked clothing) damages the skin and greatly enhances absorption (Garnier 1995).

Symptoms of poisoning with diquat or paraquat (Ellenhorn et al. 1997) are:

- early after ingestion: lesions and pains in the mouth and stomach, nausea, vomiting, diarrhoea, blood in faeces
- 48–72 hours after exposure (by ingestion, inhalation or dermally): reduced urine volume, jaundice,

12 At banana and palm oil farms, all paraquat users used gloves, aprons, respirators and boots when they loaded and sprayed paraquat. At coffee farms, use of most types of PPE was low, with the exception of the use of coveralls (48.7%). aprons (48.7%) and boots (100%).

cough, difficulty in breathing (high frequency), lung oedema (swelling), convulsions, coma.

Severity of poisoning can be distinguished as hyperacute after ingestion of massive amounts (the patients usually die after less than 4 days), acute after ingestion of 30–50mg/kg bw and subacute with usual recovery after ingestion of lower doses (Bismuth et al. 1995). Kidney failure and severe lung damage (pulmonary fibrosis) develop over several days, leading to a lack of oxygen. Death frequently occurs within one to two (and up to six) weeks and mortality is very high (see Figure 2).

Peiró et al. (2007) report one case of severe liver injury by paraquat/diquat without coexisting lung and kidney toxicity and as a consequence of environmental intact skin exposure.

A young Spanish worker who applied paraquat developed a severe dermatitis, but continued to spray. He was admitted to a hospital because of breathlessness and high fever. A liver biopsy showed centrilobular cholestasis, mild hepatocellular necrosis, and macrophagic infiltration of portal areas (Bataller et al. 2000).

7. 1. 1 Reports of skin or eye damage and systemic poisoning

Asia

In Malaysia six female plantation workers who had low cholinesterase activities in blood samples were medically examined. Three workers had itching skin or eczema or (diagnosed as contact dermatitis possibly due to pesticide), three reported having occasional pain in the chest, chest tightness and/or difficulty in breathing. Three had nosebleed (occasionally or recently). Giddiness, numbness of hands, headache, abdominal cramp, blackout, nausea and vomiting were sporadic symptoms. Five of the workers sprayed paraquat, besides other compounds. (Tenaganita & PANAP 2002).

Also in Malaysia (in 1997–1998) paraquat caused a greater proportion (19%) of occupational poisonings than organophosphates (16%) (Sirajuddin et al. 2001). In 1987 (1988) among 225 (249) pesticides identified in poisonings, paraquat was the causal agent in 62% (71%) of the total, while organophosphates were identified in 17% (14%) of cases (ibid.).

Eleven out of 27 Malaysian workers spraying paraquat (0.5% and 0.25% solutions) had one or more incidents of skin irritation or rash, mostly on the hands, legs, and in the groin or on buttocks (due to leaking equipment); one worker was injured in the eye (Howard et al. 1981).

Another study in Malaysia with 30 workers who sprayed paraquat (0.05% solution) continually over 12 weeks found that about half of the workers had irritation of the eyes (from splashes) and skin at some time. Two workers had nosebleeds and there were two cases of scrotal dermatitis (following contamination of trousers and prolonged contact) (Swan 1969).

In Sri Lanka a larger proportion of 85 spray operators (23.6%) had more skin damages than unexposed factory workers (11.8%) or general workers (15.2%). Incidence of eye damage was similar in spray men and general workers but not reported by factory workers. Nosebleeds occurred in three spray men and one factory worker but not among general workers (Senanayake et al. 1993). In the latter study the concentration of paraquat was very low (0.04–0.07%) and the workers practised excellent personal hygiene (washing frequently throughout the day); this explained the lower incidence of damage to skin and nails than reported in other studies (Senanayake et al. 1993).

These studies in Sri Lanka and Malaysia may not have observed symptoms of acute systemic poisoning. But they show the occurrence of severe irritating effects, leading to skin damage that is likely to increase the risk of paraquat absorption significantly. Localised irritant effects to skin and mucous membranes, nosebleed, cough, headache or nail damage resulting from paraquat all indicate overexposure. They should be enough to remove a worker from the area to prevent further overexposure (Zenz 1994).

Latin America

The use of pesticides is high in Costa Rica because of banana cultivation. About 175,000 workers were found to be exposed to paraquat and diquat (Partanen et al. 2003). In 2001, in 127 cases of 544 notified pesticide poisonings, the most identified causal agent was paraquat. The paraquat poisonings occurred under the following circumstances: 57 rated as suicidal,

29 accidents during work, 24 unknown circumstances, and 17 due to occupational exposure (OPS/OMS 2002a). Between 1996 and 2001 in Costa Rica paraquat was the cause of 35% of all notified poisonings (OPS/OMS 2002b). Reporting by the national surveillance system was incomplete; a study in four Costa Rican districts estimated that between 82.2 and 97.8% of pesticide poisonings were not registered. When these cases were included the proportion of poisonings in an occupational setting was 76.8% (OPS/OMS 2002c). In the banana–growing areas most injuries occurred among paraquat users, 60% of the injured users had skin burns or dermatitis, and 26% had chemical eye injuries. The remaining 14% represented systemic poisonings, nosebleeds, and nail damage (Wesseling et al. 2001a, Wesseling et al. 2001b).

Also in Costa Rica (in 1996) occupational exposure accounted for 38.5% of 1,274 pesticide poisonings registered at the national poison control centre, followed by accidental exposure (33.8%) and suicidal ingestion (22.5%). Organophosphates, carbamates and paraquat accounted for 46% of cases, with paraquat the individual agent responsible for the highest percentage of cases (11.6%) (Leveridge 1998).

The average annual rate of hospitalisations in Costa Rica due to pesticide poisoning was found to be between 115 and 130 per 100,000 workers among agricultural workers. Paraquat was the most identified pesticide causing severe poisonings, hospitalisations or fatalities (Wesseling et al. 1993).

A survey of 96 families in 1998 in a rural area of Honduras found over 80% used pesticides and paraquat was used most often. Safety measures were rare. All workers who used paraquat had at least one symptom potentially related to paraquat exposure, and prevalence of heath problems among children was abnormally high compared with national rates (Cantor & YoungHolt 2002). Paraquat poisoning has also been a major problem in Ecuador (Sevilla 1990).

United States

Between 1971 and 1985 in California 231 cases of illness due to paraquat were reported; the majority of cases (38.5%) associated with paraquat were systemic (with symptoms of acute poisoning and respiratory symptoms). Eye and skin illnesses occurred in 32% and 26% of cases, respectively, and local respiratory symptoms accounted for 3.5% of cases; 55 of the 231 cases were associated with loss of workdays and 11 cases were hospitalised (Weinbaum et al. 1995).

Also in California, (1998 to 2000), 15 agricultural poisonings with paraquat were reported. Ten of these cases were rated as definite or probable (1 with systemic and respiratory effects, 4 with eye effects, 5 with skin effects), five were rated as possible. In 2001 there were 4 poisonings reported due to paraquat, 2 cases with systemic/respiratory effects (both definite/probable) and 2 cases with localised (topical) effects (involving eyes and/or skin, one definite/probable and one possible case). Three poisonings due to paraquat were reported in 2002 with topical effects (two definite/probable cases and one possible) and in 2003, 4 poisonings were reported, 3 with systemic/respiratory effects (two definite/probable, one possible) and one definite/probable case with topical effects (CDPR 1998–2003).

Europe

After skin absorption of paraquat a worker

Figure 7: Chronic dermatitis on a leg of a female plantation worker in Malaysia, who mainly used paraquat. © PAN Asia & the Pacific.

suffered poisoning and prolonged damage to the gall (Bataller et al. 2000). In Italy, paraquat was among six pesticides most frequently associated with non–fatal poisonings referred to the main poison centre in 2000–2001; 46 poisonings out of 872 were due to paraquat (Davanzo et al. 2004).

In Crete (Greece), pesticide poisonings increased during 1991–2001 to 1,700 cases (fatal and non–fatal) per year, with organosphates and paraquat causing concern; 45% of the cases were accidental, 40% occupational and 12% suicidal (Bertsias et al. 2004). One worker was acutely poisoned by paraquat absorbed through skin during spraying (Bertsias et al. 2004). Another developed fibrosis of the lungs due to paraquat poisoning by absorption via skin; he survived with residual lung fibrosis (Papiris et al. 1995).

Among 274 fruit growers in Scandinavia, where paraquat was the secondmost used pesticide, 41% developed coughs with expectoration, 37% headaches, 30% nose discharge, 25% langour (weariness), 25% general malaise, and 21% breathlessness. Also various symptoms such as dizziness, palpitations, nausea, skin complaints or itching of the skin or eyes. A protective mask was used by 39% of the growers (Lings 1982). Among a subgroup of 181 fruit growers who were examined medically, those who used paraquat (62.4%) had lung symptoms more frequently (not statistically significant): coughing and breathlessness. It was concluded that the professional use of biocides can give rise to lung disease comprising pneumonia and chronic progressive lung fibrosis (Lings 1982). After applying paraquat another worker developed tiredness, mild breathing distress, swollen ankles and anaemia, and decreased diffusing capacity of the lungs and nephritis an inflammatory impairment of the kidney (Stratta et al. 1988).

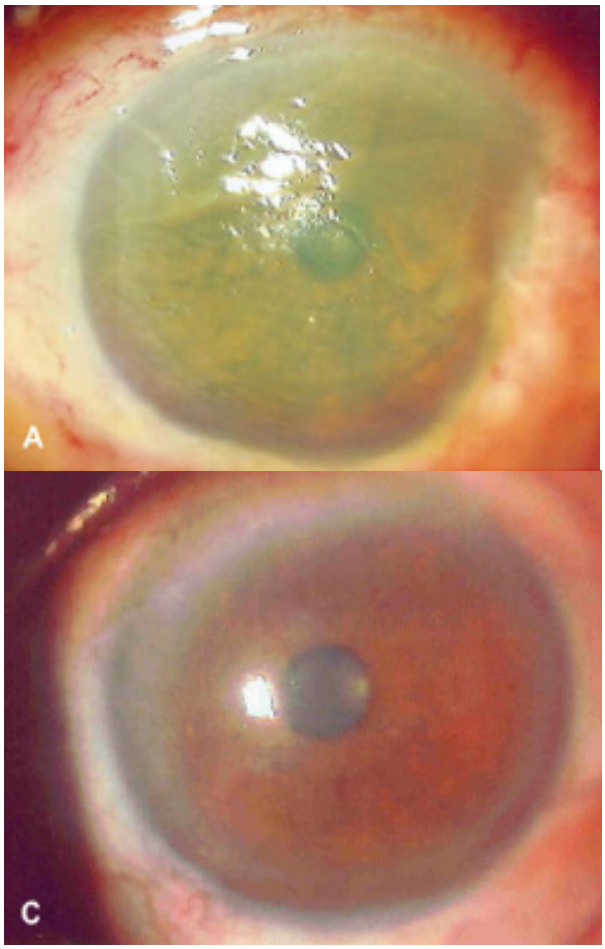

Figure 8: A–Eye injury caused by Gramoxone before treatment. C–Same eye after membrane transplantation. A haze remains. Yoon et al. (2009) © 2009 Lippincott Williams & Wilkins

In the UK between 1981 and 1986 paraquat accounted for 26 admissions to the poison treatment centre in Edinburgh; two of these occurred as a consequence of occupational exposure (leaking back canister; inhalation during spraying) and one case was due to accidental ingestion (removal of the bottle top with teeth) (Proudfoot & Dougall 1988).

Acute poisoning by inhalation of paraquat has been documented in greenhouses. A study found that 'stronger than usual solution'led to transitory failure of kidneys (Malone et al. 1971). Application of paraquat by air has caused respiratory symptoms. Depending on the sprayer type, the sizes of spray droplets could have been relatively small and may have decreased further during drift (Ames et al. 1993).

The symptoms cited in this section are an indication that work practice should be reviewed (IPCS 1984). They explain the need for strict personal hygiene and rigourous adherence to required handling procedures (IPCS 1991). However, in many countries this may represent an ideal guideline that only a minority of workers is able to follow, as it is not feasible due to inadequate conditions in the field or the hot climate.

7. 1. 2 Skin and eyes

Paraquat acts as a strong irritant, especially

in concentrated formulations, Contact with skin causes redness, blistering or ulceration and can lead to dermatitis. Diluted paraquat can cause irritation after prolonged exposure through soaked clothes (Bismuth et al. 1995).

When skin is intact, the absorption of paraquat is generally low. But it is greatly enhanced when skin is damaged. Prolonged contact with paraquat solution may itself damage the skin and allow increased absorption, leading potentially to severe poisoning (Garnier 1995). The single exposure of healthy skin to paraquat solutions has been reported to cause local lesions in skin but no systemic effect.

Among 15 cases of accidental exposure to paraquat solutions, skin burns (grade I to III) occurred in six cases, vesicles in 4 cases and contact dermatitis in one case. In 2 two cases where the face was exposed the worker suffered from conjunctivitis (Hoffer & Taitelman 1989).

Prolonged exposure to solutions containing more than 5% paraquat might lead to fatal poisoning. Exposure to less concentrated solutions may also be fatal if there are pre–existing skin lesions and if the skin is not washed immediately after exposure, or if contaminated clothing is not changed immediately (Winchester 1995; Smith 1988). The poisoning symptoms following skin absorption of paraquat are similar to symptoms after ingestion, except for local effects to the skin (Garnier 1995). Paraquat may cause contact dermatitis (Villaplana et al. 1993; Botella et al. 1985, Horiuchi et al. 2008)while diluted solutions can cause severe skin burns (Ronnen et al. 1995). Burns must be treated or else the risk of skin absorption may be increased.

Paraquat has a skin notation (IPCS 2001a; NIOSH 1996), signifying that uptake via unbroken skin can contribute substantially to total body burden and can cause serious systemic health effects (Semple 2004).

Eye contact with paraquat solution may lead to an inflammation of the cornea. Treatment usually results in recovery after prolonged healing but is not always complete and vision can be impaired if patients wait too long (Bismuth et al. 1995). Other consequences of eye contact can be conjunctivitis, an irritant inflammation of conjunctivae, and longlasting or permanent opacity of the cornea (Mc Keag et al. 2002; Ellenhorn et al. 1997). Yoon et al. (2009) report 20 cases (26 eyes) in South Korea injured by splashing with Gramoxone (containing paraquat dichloride) that were irrigated with water immediately. The grade of ocular surface injury was mild in 19 eyes (73.1%), moderate in 5 eyes (19.2%), and severe in 2 eyes (7.7%), and the mean epithelial defect

Box 5: The unkown magnitude of occupational paraquat poisonings

Estimates of the magnitude of acute paraquat poisoning are very unreliable and difficult to obtain, because it is not easy to identify paraquat as a poisoning cause. In contrast to poisonings with organophosphates, which are mostly accompanied by characteristic symptoms, the acute symptoms of paraquat poisoning may disappear intermittently and clinical effects can be delayed (Ballantyne et al. 1995).

More than 50% of the countries in Latin America, Africa, Asia and Western Pacific do not have poison centre facilities (Laborde 2004). In those countries and/or where medical services are lacking, paraquat poisoning is under-diagnosed. Especially in the rural areas of developing countries, pesticide poisoning may be frequent but often not reported. In addition, quantitative measurements in blood or urine to verify a paraquat poisoning require more sophisticated analytical methods, such as gas-chromatography (GC) plus multispectral analyses (MS) (Wang et al. 2011, Taylor et al. 2001). These type of equipment and the qualified personnel to use it, is probable not available in many countries.

In illness-surveillance report, occupational poisonings are underreported and suicides are generally over-represented (Murray et al. 2002; London & Bailie 2001). Ineffective surveillance of pesticide poisonings (IFCS 2003), and underreporting may lead to a wrong estimation of the number of illness and injury caused by pesticides (Ballard & Calvert 2001) including paraquat.

area was 63.34 ±26.67 mm^2 (range, 13.00–105.14 mm^2). Twelve patients (14 eyes) underwent amniotic membrane transplantation combined with medical treatment and 8 patients (12 eyes) received medical treatment only.

7. 1. 3 Acute respiratory effects (lung)

After absorption of a large quantity (ca. 30 mg/kg b.w.) of paraquat dichloride by any route pulmonary fibrosis develops. This pathological thickening of connective tissue in the lungs leads to a decrease in the diffusing capacity of carbon monoxide in the alveoli that can be detected from the first day. This leads to interstitial fibrosis (thickening of tissue between alveoli) and inflammation of alveoli, causing lack of oxygen, frequently resulting in death after a few days to several weeks (Bismuth et al. 1995).

Abnormalities in the lung may not be detected on chest x-rays at an early stage. But images become patchy later on. Testing lung functions can be used for a diagnosis before the stage of decreased oxygen levels is reached (Bismuth et al. 1995). If doses below 30 mg/kg b.w. are absorbed, pulmonary fibrosis rarely becomes clinically severe, and recovery of the lung function is usual. In some cases a restrictive dysfunction of the lung persists. Impairments may improve over several years (Bismuth & Hall 1995).

But in follow-up studies of survivors of paraquat poisoning, total lung capacity was significantly decreased (Yamashita et al. 2000). The destructive effects on lung tissue are a consequence of paraquat being accumulated in epithelial (tissue) cells of alveoli.

Paraquat and diquat differ in the mechanism of toxicity. Diquat is not accumulated in the lung and does not lead to pulmonary fibrosis (Rose & Smith 1977). Paraquat damages the cell membranes (lipids) by peroxidation. Levels of important enzymes are decreased, followed by an inflammatory response (Lewis & Nemery 1995). Lipid peroxidation has been associated with chronic obstructive pulmonary disease (COPD) (Santus et al. 2004).

Exposure to paraquat was associated with a higher risk for chronic bronchitis in Colombia (Arroyave 1993). Levels of antioxidants in blood samples of pesticide sprayers were increased, indicating oxidative stress (Prakasam et al. 2001). In farmers the risk of respiratory disease and mortality due to this is significantly increased. Rhinitis (inflammation of tissue in the nose) can also be caused by paraquat (ATS 1998).

7. 2 Fatal unintentional poisonings with paraquat

'One man died of unintentional paraquat ingestion. This tragedy resulted from multiple violations of pesticide safety regulations. Investigators determined that the victim was not licensed to purchase or possess paraquat. (...) The employer (...) assigned workers to apply pesticides without the training, information, or facilities that regulations require. (...) If he [the descendant] had received the prescribed training, he may not have brought a dangerous product home where he lived with his family. Most crucially, he would have learned how absolutely unacceptable it is to place any pesticide into a container that does not fully identify the contents, much less to pour it into a coffee cup as he did. Predictably, he took a sip from that cup, and although he spat it out immediately and went to the hospital about an hour later, efforts to save his life were unsuccessful.' (CDPR 2005)

Fatal unintentional poisonings have been linked with accidental intake and inappropriate behaviour, namely insufficiently diluted paraquat combined with leaking sprayers, which may lead to prolonged skin contact, severe skin lesions and paraquat absorption via skin (IPCS 1991). A number of poisonings with diluted spray solutions containing paraquat have been described. The presence of scratches to skin or small ulcers can be enough to result in absorption of a fatal dose of paraquat from the diluted spray solution.

However, fatal unintentional poisoning has resulted from the accidental contamination of the body with paraquat (20%) (Waight 1979), from swallowing a mouthful of paraquat concentrate (due to confusion of bottles), and from a smaller amount ingested (Wesseling et al. 1997). Workers died after accidentally having ingested a mouthful or sip of paraquat; in one of these cases poisoning occurred during the decanting of the concentrate (Cassidy & Tracy 2005; Ochoa Gomez & Gil Paraiso 1993).

Three fatal poisonings were caused by

accidental ingestion of diluted solutions of paraquat when workers sucked on a blocked sprayer jet (Fitzgerald 1978). Drinking from an empty bottle of Gramoxone after refilling it with water was fatal (Fernando et al. 1990). A worker who spilled a mixture of diluted paraquat and 2,4–D (in WHO class II) on the face and mouth which would appear to lead to the ingestion of a very small amount of paraquat died from acute pulmonary failure, typical for paraquat poisoning (Wesseling et al. 1997).

A review of 12 unintentional fatal poisonings resulting from skin exposure that were reported between 1974 and 1988 concluded that prolonged skin contact with paraquat solutions at concentrations as low as 5% (cation weight per volume) can cause systemic poisoning that may be fatal. It was recommended that paraquat labels should contain a warning against the use of this herbicide in knapsack sprayers (Smith 1988). Fatal poisonings have occurred following the exposure to diluted paraquat spray with much lower concentrations (see below).

Among several work related fatalities following dermal exposure to diluted paraquat, three deaths were due to a leaking sprayer (one of the workers also had dermatitis) (Athanaselis et al. 1983; Wohlfahrt 1982; Fitzgerald et al. 1978). Another two deaths occurred when the head and mouth of one worker and the back of another were accidentally contaminated during spraying (Wohlfahrt 1982).

Asia

In Japan out of 346 pesticide poisonings (90% of these systemic) that were recorded during 1998 to 2002 in several hospitals, 25% of cases proved fatal. Of these 346 cases, 36% were due to organophosphates and 20% to paraquat and diquat (Nagami et al. 2005); 65 cases (18.8% of the total) occurred during spraying, preparation, settlement, or reentry during spraying (Nagami et al. 2005).

In the Philippines two workers were hospitalised after spraying paraquat and one of them died (Quijano 2002). Two deaths occurred as a consequence of skin exposure to insufficiently diluted paraquat solutions (5% and 2.8%) and as spraying equipment was leaking (Levin et al. 1979; Jaros 1978).

In Thailand a worker who had sprayed paraquat during three months developed skin burns; he died after three more months of spraying (IPM Danida 2003).

A woman who applied paraquat appropriately diluted contaminated the scratches she had on arms and legs from branches (she had worn no protection and did not shower after spraying). Later the woman developed headaches, breathlessness, skin lesions and died several weeks after from respiratory failure (Newhouse et al. 1978). Three fatal poisonings following skin absorption occurred in Papua New Guinea. It was stated that many other cases of paraquat poisoning had not been recorded as reporting systems were inadequate (Wohlfahrt 1981).

Europe

Among 11 paraquat poisonings in Crete that were reviewed by a poison centre, 5 were fatal; six of the 11 cases were suicidal, four accidental and one occupational (Bertsias et al. 2004). In Spain a survey of data on 184 deaths by pesticide poisoning, between 1991 and 1996, found that organophosphates and carbamates accounted for most cases, followed by endosulfan and paraquat (identified as the causal agent in 11.5% of fatal poisonings) (Garcia–Repetto et al. 1998).

Costa Rica

Between 1996 and 2001 in Costa Rica 133 deaths from pesticide poisoning were registered. Of these deaths, 112 were classified as suicides, 9 as non–occupational accidents, 3 from occupational exposure; for 9 deaths the circumstances were not established. Paraquat caused 68% of all deaths and 72% of 86 suicides where the pesticide was identified (OPS/OMS 2002b). A study of occupational fatalities in Costa Rica revealed that three deaths occurred as a consequence of the exposure to diluted paraquat solution. The death of a child worker who entered a recently sprayed plantation may have arisen from absorption of diluted paraquat spray through skin and the mouth (pre–existing small ulcers on his leg would have facilitated absorption, and possibly he chewed sprayed leaves). Two deaths occurred after diluted paraquat solution was absorbed through skin only in one of these cases systemic poisoning was delayed and in the other the backpack containing the solution was leaking (Wesseling et al. 1997).

In two fatal cases the route of absorption could not be identified the suggestion was made that spray droplets could have been inhaled (Wesseling et al. 1997). A possible absorption route could be the ingestion of airborne spray solution when the worker changed from nose breathing to mouth breathing, which occurs normally during physical exertion (Frumkin 2000). The spraying of paraquat in a greenhouse has resulted in fatal poisoning (with characteristic features of kidney failure and lung injury) (Kishimoto et al. 1998). This case indicates that in certain situations the exposure by inhalation may be sufficiently high to cause poisoning.

A worker who suffered severe burns after a plane crash during the aerial application of paraquat and whose skin had been exposed to paraquat over a long period died from paraquat poisoning (Gear 2001).

8. Suicides with paraquat

'*Late last year in a hospital (...), there was a 16–year old girl who had swallowed a mouthful of paraquat immediately following an argument with her parents. The paraquat had been stored inside her house. She was cyanosed and apparently within hours or days of death, having suffered a fortnight of steadily increasing breathlessness. She could not eat or sleep because of dyspnoea, and even had difficulty drinking. She was frightened and no longer wished to die, if indeed she had ever wanted to*' (Dawson & Buckley 2007).

Gunnell et al. (2007a) estimate that there are 258,234 (plausible range 233,997 to 325,907) deaths from pesticide self-poisoning worldwide each year, accounting for 30% (range 27% to 37%) of suicides globally. The real numbers will probably never be clear, because poisoning reports are commonly based statistics of on hospital or poison centers, and people who die 'secretly' at home are usually not covered.

In general, it is difficult to distinguish between suicides and accidents (Brook 1974). In Costa Rica deaths were obviously mis–classified in several cases (Wesseling et al. 1993). For India, it has been suggested that the annual suicide rate (all types) could be six to nine times the official rate (Vijayakumar 2007).

Suicides using paraquat have been reported from the beginning of its marketing (see Hargreave et al. 1969, Fischer & Kahler 1979), and in some countries (e.g. South Korea and Sri Lanka) paraquat is the major agent used for suicide attempts. The ingestion of a lethal amount of paraquat leads to an extremely painful and prolonged death, and above a certain amount no cure helps.

Asia

In Papua New Guinea the restriction of the availability of paraquat and other toxic pesticides has been demanded because of the relatively high proportion of suicides (Mowbray 1986). Restricting the availability of paraquat was effective in reducing suicidal deaths in Western Samoa (Bowles 1995; WHO 2002).

A hospital survey in Japan involving 102 hospitaly between 1998-2004 revealed 71 cases of suicides. Of the suicides attempts with a solution with 5% paraquat and 7% diquat (n= 48) more than 80% died (n=39). All suicides attempts (n=8) with the 24% paraquat solution were fatal (Nagami et al. 2007).

Paraquat has been used for three decades in South Korea. It has caused an estimated 2,000 intoxications annually; the annual mortality among those intoxicated is 60–70% (Seok et al. 2009).

Suicides by ingestion of pesticides present a major public health problem also in Sri Lanka (Konradsen et al. 2005). Self–harm was found to be high in Sri Lanka; around 2001 organophosphates in WHO class II and paraquat accounted for the majority of poisonings (Roberts et al. 2003, Dawson et al. 2010). Mortality was high with endosulfan and paraquat, while risk factors for intentional self–poisoning were unemployment, lower educational status, problems in the family and a history of pesticide poisoning (van der Hoek et al. 2005).

Among 97 patients admitted to a hospital in the capital city Colombo in 1989 for self–poisoning only about 60% had stated that they actually wished to die and less than half (46%) knew that the agent was potentially lethal. In 59% of cases the agent was an agro–chemical, in 29% of cases it was paraquat (Hettiarachchi & Kodithuwakku 1989). However, illness

from occupational exposure to chemicals, in particular pesticide–related illness, is under reported in Sri Lanka (Kulendran 1997).

In Western Samoa, with a population of 160,000, suicide rates are 30 for every 100,000. About 80% of suicides are caused by drinking paraquat (Zinn 1995).

The Americas

In Costa Rica, paraquat was the main cause of 283 deaths due to pesticide poisoning that the Forensic Medical Department (MFD) registered between 1980 and 1987. Out of the 198 deaths where the cause was defined, 62% were suicides (Wesseling et al. 1993).

In the USA, poison centres recorded 18 deaths due to paraquat and 2 deaths due to diquat between 1983 and 1992; 15 of these 20 deaths were rated as intentional and 5 as accidental, while the majority of recorded exposures (non–fatal cases included) were accidental (Hall 1995b).

In Trinidad and Tobago, of 48 cases of suicide for the year 1996, 39 (81.3%) were due to paraquat poisoning. The incidence of paraquat-induced suicide was 8.0 per 100,000 (Hutchinson et al. 1999).

Box 6: No cure exists

The intake of a lethal amount of paraquat leads to an extremely painful and prolonged death, in some cases people suffer for several weeks (see e.g. Ong & Glew 1989).

Since, paraquat mainly accumulates in the lung (Dinis–Oliveira et al. 2008), stomach contamination using of Fuller's earth as adsorbent has not been demonstrated to be clinically effective (Pond 1995). Activated charcoal to adsorb chemicals appeared to be the best means for stomach decontamination, but no treatment has been shown to produce significant clinical benefit (Meredith & Vale 1995). Dialysis, blood filtration or fusion and antioxidants or antiinflammatory agents have not proven clinically effective to prevent a fatal outcome of serious poisonings with paraquat (Vale 2005, Bateman 2008).

Syngenta's statement 'there is an effective treatment' (Syngenta 2002, p. 27) is false and misleading.

Europe

Between 1945 and 1989 in England and Wales, 570 out of 1,012 deaths from pesticide poisoning were due to paraquat and 73% or more of these deaths were suicides (Casey & Vale 1994). In 1990 and 1991 paraquat accounted for 33 out of 44 fatalities and more than 66% were suicides (Thompson et al. 1995a). In Germany, between 1978 and 1983, 17 poison control centres (not all hospitals included) recorded 44 suicide attempts with paraquat (24 fatal), and 12 cases (2 fatal) where circumstances were not identified (Heyll 1988).

In Poland it was found that poisonings with organophosphates and bipyridylium herbicides (diquat and paraquat) were more often linked to suicide attempts than to accidents (Kotwica et al. 1997).

In Portugal, from 2000–2002, paraquat was identified as the causal agent in 31 requests for pesticide analysis at a forensic institute (mainly cases from autopsies); in 528 of the 639 requested analyses no pesticide was detected (Texeira et al. 2004).

Between 1982 to 1992 ingestion of paraquat belonged to the five most common methods of suicides in Ireland. 167 persons (42 female, 125 males) died after ingesting it (Daéid 1997).

8. 1 Suicide prevention

Paraquat was introduced to Western Samoa in 1974. Soon after, public health officials noticed a growing epidemic of self-poisoning. The total suicide rate increased from 10/100,000 to 50/100,000 in 1982. As paraquat imports fell temporarily due to financial problems, the suicide rate fell rapidly, mirroring the fall in imports (Konradsen et al. 2003). A recent study from China showed that pesticide access was a significant risk factor for suicide even after controlling for other known risk factors in social and psychiatric domains (education, living situation, marital status, income, mental disorder) (Kong & Zhang 2010).

It is widely acknowledged that the restriction of access to lethal means reduces suicides rates (Daigle 2005). This has been proved for firearms (Lubin et al. 2010; Lennaars et al. 2003), drugs, bridges, toxic domestic gas (see Daigle 2005 for numerous references) and pesticides (Gunnell et al. 2007b, Dawson et al.

2010). Figure 9 shows that the prohibition of all WHO I Class pesticides reduces the suicide rates in Sri Lanka regardless of other factors.

Banning certain pesticides is the most effective and cost-efficient measure to reduce suicides. '*Targeted pesticide restrictions in Sri Lanka over the last 20 years have reduced pesticide deaths by 50% without decreasing agricultural output*' (Dawson et al. 2010). The authors conclude that a ban of paraquat, dimethoate, fenthion in Sri Lanka would to lead to a further 33% to 65% reduction in fatalities from acute poisoning.

Many people believe that suicidal person simply change from one lethal 'instrument' to another, if one becomes unavailable, but this is not true (see Daigle 2005). People are often not so serious about committing suicide, specifically adolescents display a more impulsive behavior. Most of the people to not intend to die, but to change their lifes–and so set a signal (Meister 2011[13], Parellada et al. 2008). Owens et al. (2005) showed that of 976 persons, which attempted to commit suicides by self-poisoning, only 3.5% committed suicide in the 16 years after the first attempt. A similar earlier study, following up upon 11,583 patients over an average of 11 years showed that only 2.6% committed suicide (Zahl & Hawton 2004).

While a certain percentage of people repeat episodes of deliberate self-harm (DSH), they commonly end not fatal, despite an assumed 'learning' progress. Interestingly, persons which attempted to commit suicides by self-poisoning repeat further attempts less frequently than those cutting themselves (Lilley et al. 2008).

The access to lethal pesticides such a paraquat, can reduce the suicide rates significantly at low costs and immediately. However, other prevention measures should be supplemented:

- Education and Awareness Programs
- Screening for Individuals at High Risk
- Treatment (Therapy, follow up care for attempts)
- Media Reporting Guidelines for Suicide (Mann et al. 2005).

Locking away pesticides has not proven to be efficient on family farm level. In Sri Lanka, of 172 households that received an inhouse storage box changed the location of pesticide storage from their fields to their homes,

13 Barbara Meister, Swiss Expert on Suicide Prevention cited in Beobachter Nr. 5. 2011 pg. 61.

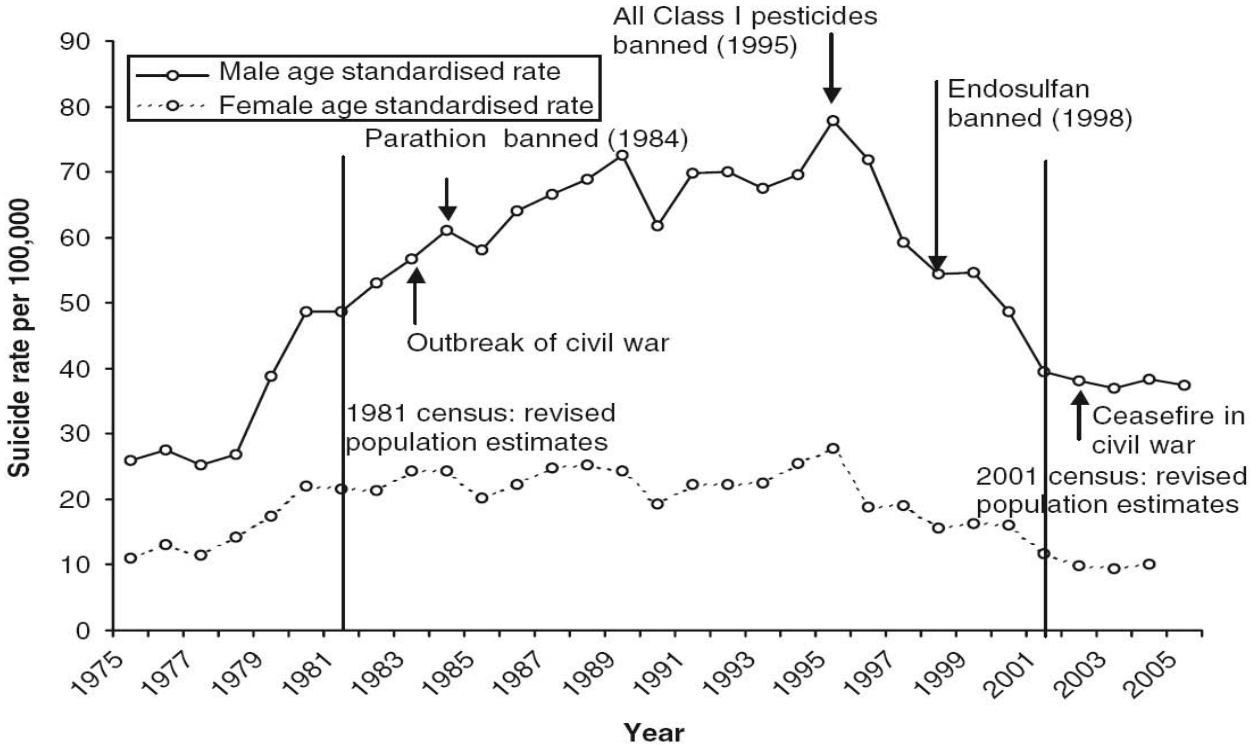

Figure 9: Age standardized suicide rates for males and females, Sri Lanka 1975–2005 (Gunnell et al. 2007b)

increasing accessibility and attention to them. Only 84% locked the box (Konradsen et al. 2007). Syngenta lowered the amount of paraquat in one formulation (INTEON) to ca. 30% and changed the formulation, but this showed no significant benefits (Wilks et al. 2011, Bateman 2008). Already in 2000, Chishiro (2000) found out that the decrease of paraquat concentration does not reduce the number of fatalities. In Japan, of 49 persons poisoned with a mixture of 'only' 5% paraquat and 7% diquat 36 died (Nagami et al. 2005, see also Nagami et al. 2007). This is a case fatality ratio of 73%.

9. Chronic health effects of paraquat

Exposure to relatively low doses of paraquat but over a longer period of time can affect the lungs, nerve system, brain, skin and eyes. Just over 30% of fruit farmers in Taiwan had dermatitis of the hand, more often on the right hand (Guo et al. 1996). Half the farmers used paraquat. Contact dermatitis is a significant health problem for banana workers in Panama, who are exposed to paraquat (Penagos 2002). This condition increases the risk for skin absorption. In epidemiological studies the long–term exposure to low doses of paraquat was linked to small changes in gas exchange of the lung and was associated with an increased risk of developing Parkinson's disease.

Despite treatment incl. membrane transplantations, accidental splashes of Gramoxone (containg paraquat dichloride) have lead to persistent sight impairments (see Yoon et al. 2009 above).

Chronic exposure can affect reproduction. Birth defects may result. The exposure of male workers to paraquat and diquat was associated with a relative risk of 2.77 (95% confidence interval 1.19–6.44) of congenital malformations and birth defects occurring in their children (Garcia et al. 1998). Paraquat was found to adversely affect embryonal development in animal tests (Hausburg et al. 2005).

9. 1 Chronic respiratory effects (lung)

In test animals, the repeated exposure to small quantities of paraquat in diet or via skin can cause pulmonary fibrosis, and exposure to respirable size droplets caused direct injury to the lung (Bismuth et al. 1995). Droplets of a respirable size have an increased toxicity to the lung but most sprayer types produce droplets that are too large to enter the alveoli. But irritant effects on the upper airway are common (Hall & Becker 1995).

Chronic exposure of workers to paraquat and potential impacts on the lung have been the subject of several studies. Two studies found no association of paraquat exposure with respiratory effects, while three others observed a positive association with small alterations in gas exchange (see Chapter 7.1.3).

Damages to the lung cannot always be recognised in chest x-rays or respiratory tests at an early to intermediate stage (Bismuth et al. 1995; Vale et al. 1987). Evaluating total lung capacity (from a single breath) and measuring diffusion capacity (for carbon monoxide) are more sensitive methods than spirometric tests to assess potential restrictive lung conditions (ATS & ERS 2000). Measurement of oxygen uptake during maximum exercise further increases the sensitivity (Schenker et al. 2004). In tests on rats exposure via the skin to repeated doses of paraquat solution (0.8–2.85%) led to an increase in the thickness of lung arteries and hemorrhage (Levin et al. 1979).

9. 1. 1 Studies of chronic effects on the lung

A WHO study identified paraquat as among the pesticides with a priority for further examination due to its wide use and numerous severe and fatal poisonings (WHO & UNEP 1990). While many cases were accidental, acute poisoning with paraquat is characterised by delayed pulmonary fibrosis, and it could not be excluded that chronic exposure to low (non–fatal) doses could have an influence on the lung function (WHO & UNEP 1990).

In studies with plantation workers who had sprayed paraquat over longer periods of time, it was concluded that the long term occupational use of paraquat is not associated with lung damage or adverse effects in exposed workers (Senanayake et al. 1993).

Methods for medical examination in these studies (chest x-rays and spirometric tests of lung function) were insufficient to diagnose paraquat poisoning, except for measurement of the diffusion capacity of carbon monoxide. Two other studies with workers who had sprayed paraquat over a longer period concluded that working with paraquat under field conditions is associated with de–saturation of arterial oxygen during maximum exercise in a dose dependent fashion (Dalvie et al. 1999), and that the increased prevalence of respiratory symptoms in the exposed workers suggested an effect of long-term paraquat exposure on respiratory health (Castro–Gutiérrez et al. 1997).

The hypothesis presented was that subacute exposure to paraquat (to lower doses, possibly over a longer time) may lead to decreased diffusing capacity, and that lung fibrosis is not caused except in cases of an acute and substantial exposure (Castro–Gutiérrez et al. 1997; and reference therein: Levin et al. 1979). In more intensely exposed workers the relative risk for chronic bronchitis was twice as high (not statistically significant), while for episodic shortness of breath accompanied by wheezing it was 2.9 (95% confidence interval 1.46.3) (Castro–Gutiérrez et al. 1997).

A third study with 338 workers from plantations in Costa Rica found that paraquat exposure was associated with small but statistically significant changes in gas exchange in the lung. Levels of exposure could be different on small farms with fewer workers; these were not included in the study. Ventilatory equivalent for CO_2 (respired air volume for uptake of certain amount of oxygen), arterial oxygen de–saturation (difference between oxygen saturation of blood at rest and maximum exercise) and carbon monoxide diffusion capacity were measured, the lung function was tested and cumulative exposure to paraquat was estimated for individual workers. The diffusion capacity and lung function in spirometric tests did not differ between paraquat handlers and non–handlers, and no clinically significant increases in restrictive lung disease or interstitial thickening were observed. Cumulative exposure to paraquat was associated with an increased relative risk for chronic cough of 1.8 (95% confidence interval (CI) 1.0–3.1) and with an increased relative risk for shortness of breath accompanied by wheeze of 2.3 (95% CI 1.2–5.1). Cumulated paraquat exposure was associated with an increase in the ventilatory equivalent for CO_2 in a statistically significant manner (this factor accounted for a small portion of total variance); paraquat exposure was associated with oxygen de–saturation (5% or more) with a relative risk of 1.7 (95% CI 0.9–3.0) (Schenker et al. 2004). The latter findings suggest that exposure to paraquat may be associated with subclinical abnormalities in gas exchange of the lung (Schenker et al. 2004; Dalvie et al. 2005).

Farmers (non–asthmatics) in the US who used paraquat had over a threefold relative risk for wheeze (whistling in the chest). When asthmatics were included the risk increased by 27%, a significant rise (Hoppin et al. 2002). Nine workers in South African vineyards whose trousers had been soaked with paraquat

Box 7: Compensation for diseases caused by paraquat

The use of paraquat under the normal prevailing conditions may result in acute and chronic poisoning. Plantation owners profiting from this situation should be fully liable for these consequences. Pesticide manufacturer share the responsibility for health effects if they market pesticides in countries where usual working conditions result in high risks for a large proportion of users (see FAO Code of Conduct Article 5.2 [FAO 2002a]).

Plantation workers even in developing countries thus can claim that employers and/or manufacturers of toxic products in industrial countries are liable for negligence for intentionally exposing them to a toxic substance.

In 2007, for example, the Los Angeles Superior Court awarded $3.3 million to six Nicaraguan farmworkers who had sued Dole Food Company in the US arguing they had been rendered sterile some three decades ago by the international corporate giant's application of DBCP, a pesticide proven highly toxic and banned in the US, on the plantations where they worked on (Rosencranz et al. 2009).

spray developed redness and burning of legs. For six of these workers diffusion in the lung of carbon monoxide was reduced, while two of the workers reported chronic coughing and expectoration,, and one had difficulty in breathing (Levin et al. 1979).

In Antioquia, Colombia, 11% of 5,483 people interviewed in 1986 used paraquat (15.2% of the rural and 4.4% of urban population), normally with knapsack sprayers. 17% reported having experienced illness during the 2 weeks before the study and 7.2% of the problems were related to the respiratory system (mostly coughing, runny nose, expectoration, dyspnea or shortness of breathing). 62.5% of participants had the problems for less than 15 days, 22.7% between 2 and 12 weeks and 10.1% for at least 1 year. A sub–sample of 896 people was medically examined and a physician diagnosed chronic bronchitis (accounting for 12.8% of the effects), asthma (2.7%) and tuberculosis (0.2%). In the sub–sample the relative risk for chronic obstructive pulmonary disease in paraquat users was three times higher than in nonusers and the association was highly significant for smokers, indicating a combined effect. Chronic bronchitis was more prevalent among paraquat users than nonusers in smokers and nonsmokers (Arroyave 1990).

In a follow-up study in the same area with 1,157 children of paraquat users, exposure to paraquat was associated with the incidence of chest colds. The relative risk was almost three times higher in the group of children with a high level of paraquat exposure, and increased by a factor two or more for the group with low and moderate levels of exposure (IDRC 2003).

9. 2 Carcinogenic potential

Tumours occurred in one out of three long-term studies with rats; the weight of evidence suggested paraquat was not carcinogenic in rats. Another conclusion was that paraquat is unlikely to pose a genotoxic risk to humans (FAO 2004). Positive test results for mutagenicity were found in human lymphocytes and lung cells of hamsters (FAO 2003).

The available evidence indicates that reactive oxygen species produced by paraquat are responsible for its genotoxicity. It was assumed that genotoxic effects will not be evident below a certain threshold concentration, provided that the antioxidant defence mechanisms of the organism have not been overwhelmed (FAO 2004). In animal studies, however, genotoxic effects of paraquat have been observed even following the absorption through skin (D'Souza et al. 2005).

In human lymphocytes (white blood cells), paraquat induced slight but significant increases in the frequency of sisterchromatid exchanges (Ribas et al. 1997–98). This indicates damage to chromosomes (structure carrying genetic information) leading to an increased susceptibility to malignant tumours (Segen 1992).

Paraquat has been rated as 'Group E--Evidence of Non-carcinogenicity for Humans' by the US Environmental Protection Agency (US EPA 2010[14]). But, based upon another study it has been rated as 'C; possible human carcinogen' because of the induction of squameous cell carcinoma in rats (US EPA 1993)[15]. Among factory workers who had manufactured 4,4'bipyridyl (a precursor used in paraquat production) the incidence of skin lesions was increased and these progressed to Bowen's disease (precancerosis of the skin) and, in fewer cases, to squameous cell carcinoma. It appears that exposure to sunlight was a cofactor and production has been modified in the meantime (Hall & Becker 1995).

Paraquat contains 4,4'bipyridyl as an impurity in concentrations of up to 0.2% (Ambrus et al. 2003). The maximum allowed concentration is 0.1% and levels were normally below 0.05% (FAO 2003b). It has not been clearly established so far whether carcinogenic effects are caused by paraquat or by related bipyridylium compounds (Li et al. 2004). A test in mouse lymphoma cells was positive with paraquat (US NTP 2005).

The risk for malignant melanoma (skin cancer) was increased among male agricultural workers exposed to paraquat. In eight out of ten cases melanoma were situated on the lower limbs, where exposure to sunlight is less plausible than skin contact with pesticides

14 The rating in US EPA 2010 is derived form a report of the Office of Pesticide Programs (OPP) dated 15th March 1989.
15 see http://www.epa.gov/iris/subst/0183.htm II. Carcinogenicity Assessment for Lifetime Exposure. Last Revised — 10/01/1993

DBCP and paraquat in particular (Wesseling et al. 1996). Total pesticide use (indexed per agricultural labourer) on coffee and banana was associated with increases in the relative risk for skin melanoma, lung and penile cancer in male workers. Paraquat is used extensively on banana and coffee. The increase could not be explained by smoking (Wesseling et al. 1999).

9. 3 Paraquat & Parkinson

9. 3. 1 The Disease

Parkinson's disease (PD) is regarded as the most common degenerative disorder of the aging brain after the Alzheimer's dementia. The incidence and prevalence of Parkinson's disease increase with advancing age, occurring in about 1% of people over the age of 65 years (WHO 2004). PD is currently still considered to be non-heritable, but most likely the disease reflects a combination of genetic susceptibility and unknown environmental factors (Westerlund et al. 2010, Lev & Melamed 2001, Ritz et al. 2009).

PD is characterized by the tetrad of tremor at rest, slowness of voluntary movements, rigidity, and postural instability (Bové et al. 2005).

The main biochemical abnormality in PD is the profound deficit in brain dopamine level attributed to the loss of neurons of the nigrostriatal dopaminergic pathway (ibid).

9. 3. 2 Paraquat and Parkinson's Disease

Numerous studies – many based on animal experiments, but also epidemiological studies – have discussed the possible role of pesticide exposure, in particular paraquat, in the risk of developing Parkinson's disease (PD) (e.g Elbaz et al. 2009, Kamel et al. 2007, Brown et al 2006, Dick 2006, Ascherio et al. 2006, Firestone et al. 2005, Petrovitch et al. 2002).

Seven studies (see Table 2) found that the risk of developing PD was higher among workers who had been exposed to paraquat. According to Ritz et al. (2009), the risk was up to 4.5 times higher for susceptible people who had been exposed both to paraquat and the fungicide maneb. This may be due to a synergistic effect of paraquat and maneb (Cory–Slechta et al. 2005). One study found no increase of risk, and another found that this was reduced (Engel et al. 2001). Liou et al. (1997) showed that workers who had used paraquat for over 20 years had an increased risk of 6.44 (95% Confidence Interval [CI] 2.41–17.2). Tanner et al. (2011) found a strong association between Parkinson's Disease and paraquat and suggested a 2.5 times increased risk. However, when people were exposed to different pesticides and other contaminants, it is more difficult to establish a statistically

Table 2: Epidemiological studies linking Parkinson's Disease to paraquat exposure

Studie and Country	Risk Ratio (95% Confidence Interval)	Comment
Hertzman et al., 1994, Canada	1.25 (0.34–4.63)	Hospital PD cases
Hertzman et al., 1994, Canada	1.11 (0.32–3.87)	Community PD cases
Seidler et al., 1996, Germany	—	
Liou et al., 1997, Taiwan	3.22 (2.41–4.31)	
Kuopio et al., 1999, Finland	1.2 (not significant)	
Engel et al., 2001, USA	0.8 (0.5–1.3)	Parkinsonism
Firestone et al., 2005, USA	1.7 (0.2–12.8)	
Kamel et al., 2007, USA	1.8 (1.0–3.4)	Prevalent PD
Kamel et al., 2007, USA	1.0 (0.5–1.4)	Incident PD
Ritz et al., 2009, USA	2.99 (0.88–10.2)	paraquat & maneb exposure
Ritz et al., 2009, USA	4.53 (1.70–12.1)	paraquat & maneb exposure susceptible persons
Tanner et al., 2009, USA	2.80 (0.81–9.72)	
Tanner et al. 2011, USA	2.5 (1.4–4.7)	
Source of Table: Berry et al. (2010). Modified and complemented by results of Tanner et al. 2009/2011 and Ritz et al. 2009		

significant association for individual pesticides.

While experiments with animals have shown that paraquat can induce Parkinson's Disease (see Chen 2010 and references therein), others raise doubts regarding paraquat's neurotoxicity because of the blood brain barrier (BBB), which prevents that toxins enter the brain (see Bartlett et al. 2009).

In vitro studies have shown that paraquat is toxic to dopamine cells, achieving in vivo brain concentrations required for toxicity has been questioned. Many rodent studies have reported the ability of systemic paraquat administration (basically the injection of paraquat) to induce parkinsonism but other scientists find the contrary (ibid.). A recent study by Rojo et al. (2007) on inhalation of paraquat in rodents showed no neurotoxicity.

The key question is how much evidence is needed to take regulatory measures? Is an association between Parkinson's Disease and paraquat identified by seven studies not enough? Paraquat is definitely not necessary enough to accept any risk. The adverse effects of asbestos dust, the ozone 'hole' created by chlorofluorocarbons (CFCs) were recognized very early, but for a long time no action was taken. In consequence, thousands of people will develop cancer (EEA 2001). Waiting for more 'evidence' might cause tremendous health costs and suffering. The European aims at a ban of all pesticide which are 'probable' human carcinogens, mutagenic and/or reproductive toxin (CMR) (EU 2009).[16] A substance should also not be approved if it has the inherent capacity to cause neurotoxic effects (ibid.). The probability or the capacity to cause adverse effects seems to be sufficient to exclude a pesticide from authorization. Paraquat's authorizations needs to be reviewed in the light of the effects on the neuronal system and the precautionary principle.

16 Active ingredients which are (or have to be) classified in accordance with Regulation (EC) No 1272/2008 in Category 1B for CMR properties.

10. Implications for wildlife and the environment

10. 1 Risks to vegetation, wildlife and soil microorganisms

The hazards of paraquat to the environment are rated in the EU as follows:

- Dangerous for the environment (Symbol N) resp. Aquatic Acute 1 & Aquatic Chronic 1;
- Very toxic to aquatic organisms (Risk Phrase 50) resp. Very toxic to aquatic life (Hazard Statement 400);
- may cause long-term adverse effects in the aquatic environment (Risk Phrase 53) resp. Very toxic to aquatic life with long lasting effects (Hazard Statement 410) (EU 2008).

Among 40 herbicides commonly used on field crops in Australia, paraquat has the highest acute toxicity (based on the acute oral LD_{50} in rats) (DPI 2004). While paraquat is not volatile as a solid, the drift of spray solutions could potentially be a problem for animals due to its toxicity (US EPA 1997). In wildlife, the sublethal effects from exposure to lower doses of pesticides can be important, as altered behaviour as a consequence of lowlevel pesticide exposure may be almost as fatal in nature as an acute lethal dose (Kjolholt 1990).

Paraquat is moderately hazardous to bird species based on LD_{50} values (Tomlin 2003) and rated by the WHO Ranking of Acute Hazard (WHO 2010). An acute LD_{50} of 35 mg/kg bw for birds signifies that paraquat can be highly hazardous to some bird species (EC 2003b).

On embryotoxicity to birds' eggs it was observed that the exposure of eggs from chicken and Japanese quail to a spray solution of 0.4% caused mortality and defects of the lung in young birds. Immersion in a 0.05% solution led to a decrease in hatching success. Paraquat appeared to be the most highly embryotoxic and teratogenic (causing malformations of an embryo or foetus) herbicide. The lethal concentration (LC_{50}) for immersion of mallard eggs in a solution was 0.18% (Hoffman 1990).

The US EPA found that its level of concern is

exceeded at recommended application rates of 1.12 kg paraquat per hectare. But it asserted that a risk to birds only exists shortly after application until spray solution has dried; it was concluded that the uses that are registered in the US are not expected to pose significant acute risk to bird species (US EPA 1997a). Regarding chronic risk to birds, the level of concern was exceeded at recommended rates. The EPA was concerned that direct use of paraquat may affect reproduction of birds but estimated that concentrations reaching eggs are not expected to be enough to cause significant mortality, or reductions in the proportion of eggs that hatched and again in the growth of birds (US EPA 1997a).

To mammals, paraquat is highly to moderately hazardous, based on WHO ranking and LD_{50} values ranging from 22 to 157 mg/kg bw (Smith & Oehme 1991). Some of the EPA's risk quotients for acute and chronic risks to mammals were exceeded but it was asserted that data on environmental fate indicate that paraquat is not available to mammals once it dries (US EPA 1997a). In the EU review for authorising paraquat, it was found that hares died and small mammals were affected, but the extent could not be estimated (EC 2002a).

Paraquat is slightly toxic to fish species based on LC_{50} values (Tomlin 2003) and narrative rating according to Kamrin (1997). It was found to be moderately hazardous to some fish species in the juvenile stage (de Silva & Ranasinghe 1989). At recommended paraquat concentrations for control of aquatic weeds (0.1–2.0 mg/l), LC_{50} values were not exceeded but toxicity was increased by erratic swimming, arhythmic heart beat or nerve pulses, gill lesions and bleeding points in the fins and tail (Tortorelli et al. 1990). In carp, paraquat accumulated in all organs studied and accumulation increased with the water temperature. Paraquat was seen to inhibit acetylcholinesterase (an enzyme that stops signalling in the nervous system). Susceptibility to infectious diseases increased with long-term exposure, indicating induced stress (Láng et al. 1997; Nemcsok et al. 1987).

At water concentrations above 0.2 mg/l, paraquat caused malformations in all frog tadpoles of a batch, whereas growth was reduced at concentrations of 0.1 mg/l and above. It was concluded that paraquat should be classified as a teratogen (Osano et al. 2002). The LC_{50} (96 hours) for frog tadpoles was 22 mg/l and changes in gill tissue were noted. Results indicated that populations of frog species could be affected by paraquat at concentrations below the LC_{50} value and pesticide use near surface waters caused concern (Lajmanovich et al. 1998).

For two species of daphnia, paraquat was moderately toxic based on median effective concentrations (EC_{50}) of 2.57 and 4.55 mg/l, respectively; chronic exposure may be dangerous for natural populations (Alberdi et al. 1996). Application of paraquat to water (at 1 mg/l) led to uptake by water snails, which contained 0.43 mg/kg (NLM 1994). It was found that rates recommended for paraquat application against aquatic weeds would affect the population growth of phytoplankton species in rivers, which would affect other species (Sáenz et al. 2001). A recommendation was made to limit the use of paraquat to water courses where it could pass easily into natural waters (Láng et al. 1997).

It has been asserted that paraquat does not bioaccumulate (Tomlin 2003).A bioconcentration factor (BCF) of 0.05–6.9 was calculated for paraquat based on studies with several fish species (NLM 1994). According to the EU a pesticide is bioaccumulative if the BCF above 2000 (EU 2009).

10. 2 Degradation of paraquat in soil and water

In certain soils paraquat is biologically inactive and is not available to plants or microorganisms. When strongly bound to soil it has no phytotoxic effects and may persist indefinitely (Mordaunt et al. 2005; Hall 1995a). Paraquat is adsorbed (held) to a greater extent by soil with high cation exchange capacity (CEC); this increases with clay content. The strong adsorption capacity (SAC), or maximum amount of paraquat that could be inactivated by a soil, was estimated to be several hundred times higher than the amount of paraquat that is normally applied during one year (Smith & Oehme 1991).

The SAC or capacity of a soil for inactivating paraquat is lower than the CEC (Damanakis 1970). For several soils the SAC was only 10–30% of overall CEC (Summers 1980). The

desorption of soilbound paraquat depends on the soil's CEC and the desorbing cation. Paraquat is slightly mobile in sandy loam soil and potentially mobile in sandy soils with extremely low organic matter content (US EPA 1988).

In the presence of other cations the desorption may potentially increase, e.g. as a consequence of salinisation in irrigated soils or fertilisation. When the calcium or sodium concentration in soil pore water increased tenfold the SAC for paraquat decreased by 17% to 40% (Kookana & Aylmore 1993). Fine clay fractions and (solid) organic matter can contribute significantly to the SAC (Hseu et al. 2003; Spark & Swift 2002).

Certain clay minerals adsorb paraquat less strongly than others. It was seen with kaolinite clay that paraquat slowly became available to plant roots and killed cucumber plants, while paraquat adsorbed on soil with 1% content of montmorillonite was not available to plants as long as the amount was below the SAC. Adsorption of paraquat on clay minerals affects their capacity for holding water or nutrient elements in a beneficial or deleterious manner (Weber & Scott 1966).

In laboratory trials, paraquat was mobile to a limited extent in a soil containing mainly kaolinite and vermiculite clay when the SAC was exceeded (at very high application rates) (Helling et al. 1971). In a field trial where paraquat had been applied at very high rates over ten years, it was found that the residues of paraquat in soil reached a maximum level and declined after some time due to degradation in soil pore water. It was concluded that under normal use (good agricultural practice) no toxic effects on the crop plants or soil organisms occur (Roberts et al. 2002). The FAO does not consider potential phytotoxicity from paraquat residues in soil to be a relevant problem (FAO 2000).

In one study, paraquat was applied to a sandy loam soil over six years at an annual rate of 4.48 kg/ha. Soil analysis after seven years revealed that essentially all of the applied paraquat was still present. A significant amount had penetrated to soil layers of 25–36 cm (probably due to a lower clay content), while most of the paraquat remained in the topmost 5 cm (Fryer et al. 1975). No significant degradation occurred (neither through light nor microorganisms).

Although paraquat residues caused no phytotoxic effects, calculations of the long-term ability of soils to inactivate paraquat should not make allowances for possible degradation unless specific information is available for the local site (Fryer et al. 1975). It was deemed unlikely that accumulation of paraquat in medium and heavy soils with a relatively high clay content would damage the crop but in lighter sandy soils loosely bound (extractable) paraquat was available to plants (Riley et al. 1976) and led to phytotoxic effects (Tucker et al. 1967). For seven different soils the estimated SAC of the top 2.5 cm layer ranged from 63 to 3228 kg/ha with median and mean values of 280 kg/ha and 889 kg/ha, respectively (Knight & Tomlinson 1967).

Soils from 20 coffee plantations had an average inactivation capacity of 0.1–0.5 g paraquat per kg of soil. Where paraquat had been applied at a rate of 2.6 kg/ha per year over 20 years, the total residues present in the soil comprised up to 10% of the soil's inactivation capacity (Constenla et al. 1990). The topmost 2.5 cm layer of these soils constitutes an inactivation capacity for paraquat of 25 to 125 kg/ha. With an annual input of paraquat as stated (2.6 kg/ha,) the inactivation capacity of the topmost 2.5 cm in the soils would be expected to be saturated after 9.6 to 48 years without degradation. Input of paraquat is very high on some sites, e.g. on banana plantations where it is sprayed monthly (OPS/OMS 2001b).

Paraquat was found to have accumulated in sediments of lakes with drainage ditches that had been treated with 1.6 kg/ha of paraquat (more on overgrown sites) each year. Suspended soil particles with adsorbed residues were transported into the lake and deposited on the ground of drainage ditches and in lake sediment. No significant degradation occurred (Betz 1975). Based on chemical extraction of bound residues, the SAC of the top 15 cm sediment layer (10% clay content) for paraquat was estimated at 182 kg/ha (or 1.07 g per kg of soil, dry weight) on average, comprising only 1.4–2.8% of the sediment's CEC (Wegmann 1977).

In biological assays the amount of paraquat in the sediment required to inhibit root growth of plants by 50% was determined as 0.73 g/kg, equivalent to 124 kg/ha (for top 15 cm

layer). This was taken to be a more realistic estimate of inactivation capacity (with significant inhibition already occurring). The inactivation capacity would be saturated by the continued input of paraquat after over 100 years at the given rate but much earlier if the rates were increased. It was recommended to discontinue application to drainage ditches over a longer period to avoid putting the ecosystem at danger sooner or later (Wegmann 1977).

In water, paraquat is adsorbed on the sediment, plants or suspended particles (Summers 1980). But paraquat in surface waters could be transported if soil particles with adsorbed paraquat are carried offsite as a consequence of erosion (US EPA 1997a). It appears that the inactivation capacity of soils could be saturated within the foreseeable future where the annual application rate of paraquat is above usual rates, or on soils with a low clay content or again where cation concentrations are high.

Fertiliser may increase the mobility of paraquat (Smith & Mayfield 1978). In loam and kaolinite soils the amount of adsorbed paraquat decreased with increasing concentrations of ammonium (Wagenet et al. 1985).

The halflife of paraquat in soil has been determined as 6.6 years (Hance et al. 1980). Depending on site conditions, degradation may proceed more rapidly or more slowly. Halflife in fields ranged up to 13 years (USDA 1995). Provided that the net input rate exceeds the net degradation rate, which appears feasible due to the very high persistence of strongly adsorbed paraquat, the capacity of any soil to inactivate paraquat will be saturated sooner or later through continued input.

In peat soils (with a high organic content) paraquat remained in a thin top layer at a high concentration and it was concluded that its application was only acceptable when it was mechanically incorporated into the soil to a depth of 610 cm (Damanakis et al. 1970). The authors stated: '*Rainfall seems unable to move paraquat into soil. After repeated applications of paraquat on an undisturbed soil, occurrence of a thin layer of high concentration of paraquat is to be expected*' (Damanakis et al. 1970). This means that the use of paraquat in notillage systems is likely to be associated with an increased risk of toxic effects on crop plants after a prolonged period of applications.

A review on the fate of paraquat in soil found that the addition of small amounts of organic matter, kaolinite, vermiculite and montmorrilonite to soil reduced the availability to plants at an increasing rate, The bioavailability of paraquat was increased by the addition of lime (Weber et al. 1993). Tropical soils are more varied in the type of clay minerals. While microbial degradation of pesticides generally proceeds at higher rates due to the higher temperature than in the temperate zone degradation rates in both zones may be comparable in the dry season (Racke et al. 1997).

Weathered kaolinite soils in the humid tropics had a decreased capacity to inactivate paraquat when compared to soils of high montmorillonit (clay) content (Wagenet et al. 1985). In organic soils the primary inactivator for diquat and paraquat is organic matter (Weber et al. 1993). Dissolved organic matter (humic acids) in soil interacts with adsorbed species including paraquat and may promote desorption following heavy rainfalls (Andersohn 2002).

In Spain surface water was analysed for bipyridylium herbicide. In a wetland, paraquat was detected in 2.4% of samples (2 out of 84), in the lagoon in 6.3% of samples (18 out of 288), while in marsh water paraquat was measured in 9.0% of samples (13 out of 144). Diquat was detected more frequently and maximum concentrations of paraquat were measured near rice fields (Fernandez et al. 1998). The average concentration of samples where paraquat was detected in was 0.78 µg/l, which is 7.8 times above the drinking water limit in the EU (0.1 µg/l), while the highest concentration was 39.5 times above the limit.

It was concluded that diquat and paraquat are ubiquitous in the Mediterranean environment and that their use on rice and other crops should be controlled (Fernández et al. 1998). Paraquat was also present in surface and groundwater in Andalusia, Spain (Vidal et al. 2004).

In St. Lucia, in the Caribbean, residues of paraquat measured in drinking water were above 0.1 pg/l in several samples, ranging up to 5.3 pg/l (Boodram 2002).

11. Regulatory controls and guidance for the users

11. 1 International standards regarding acutely toxic pesticides

At the international level the UN Food and Agriculture Organisation (FAO), the World Health Organisation and the International Labour Office (ILO) make recommendations on the distribution and use of pesticides and establish standards for workers' protection. These provide guidance to countries in the establishment of national health and safety standards. Part of international policy is based on voluntary adherence of governments, retailers, producers and industry. The FAO has made specific recommendations for the use and marketing of pesticides in the International Code of Conduct since 1986.

The central place of health in the international agenda for sustainable development is reconfirmed in the Plan of Implementation of the World Summit on Sustainable Development (September 2002), which also emphasizes the importance of the precautionary principle. It presents actions for changing un–sustainable production/consumption patterns:

- Sound management of chemicals. By 2020, aim to achieve that chemicals are used and produced in ways that lead to the minimisation of significant adverse effects on human health and the environment, using transparent science–based procedures of risk assessment and risk management, taking into account the precautionary approach. Support developing countries to strengthen their capacity for sound management of chemicals. Include action at all levels to further develop a strategic approach to international chemicals management based on the Bahia Declaration and Priorities for Action beyond 2000 of the IFCS.
- Strengthen and promote programmes of the ILO and WHO to reduce occupational deaths, injuries and illnesses, and link occupational health with public health promotion.
- Promote and improve sciencebased decisionmaking and reaffirm the precautionary approach as set out in the Rio Declaration on Environment and Development (UN DESA 2002, items 23, 23b, 54m and 109f).

The UN Commission on Human Rights has discussed issues of general and illegal traffic in toxic substances in Latin America and Africa. It found that the most serious concerns raised were in connection with the excessive or uncontrolled use of toxic agricultural products such as paraquat and dibromochloropropane (DBCP) (UNESCO 1999). Both at the national and international level there is a continuing need for regulation of the trade and use of chemicals.

In 2006 the Strategic Approach to International Chemicals Management (SAICM), an outcome of the World Summit on Sustainable Development (September 2002) was published. This global approach is needed because:

> *a.) The existing international policy framework for chemicals is not completely adequate and needs to be further strengthened;*
>
> *b.) Implementation of established international policies is uneven;*
>
> *c.) Coherence and synergies between existing institutions and processes are not completely developed and should be further improved;*
>
> *d.) There is often limited or no information on many chemicals currently in use and often limited or no access to information that already exists;*
>
> *e.) Many countries lack the capacity to manage chemicals soundly at the national, subregional, regional and global levels;*
>
> *f.) There are inadequate resources available to address chemical safety issues in many countries, particularly to bridge the widening gap between developed countries on the one hand and developing countries and countries with economies in* transition on the other.'

SAICM included three work areas with regard to pesticides and suggests 20 activities for implementation. Activity 23 is particularly

'revolutionary': '*Base national decisions on highly toxic pesticides on an evaluation of their intrinsic hazards and anticipated local exposure to them*.' (UNEP 2006). Basically, this means an acknowledgement of the user reality – not idealistic and unrealistic '*proper use conditions*' should be the base for authorization, but the '*anticipated local exposure*'.

11. 1. 1 UN Food and Agriculture Organisation; World Health Organisation

The UN Food and Agriculture Organisation (FAO) and the World Health Organisation (WHO) have recommended restrictions on availability of toxic pesticides.

Following its endorsement of SAICM (see above) the FAO Council suggested that one of the activities that FAO could focus on the reduction the risks posed by highly hazadous pesticides (HHP), including a possible progressive ban of such products.

The FAO Panel of experts on pesticide management concluded that HHPs are–among other criteria–'*pesticide formulations that have shown a high incidence of severe or irreversible adverse effects on human health or the environmen*t' (FAO/WHO 2007).

Considering the extremely large number of fatal but also serious non-fatal incidents with paraquat formulations–there is no doubt that paraquat qualifies as highly hazardous pesticide and needs to be phased out.

FAO's International Code of Conduct on the Distribution and Use of Pesticides

The FAO demanded over 20 years ago that farmers in the tropics should abstain from using pesticides that would require impractical and expensive protective equipment (FAO 1986; FAO 1990a). In the International Code of Conduct on the Distribution and Use of Pesticides and in the Provisional Guidelines on Tender Procedures for the Procurement of Pesticides, the FAO renewed these recommendations:

- Pesticides whose handling and application require the use of personal protective equipment that is uncomfortable, expensive or not readily available should be avoided, especially in the case of smalls–cale users in tropical climates (5). Preference should be given to pesticides that require inexpensive personal protective and application equipment and to procedures appropriate to the conditions under which the pesticides are to be handled and used. (FAO 2002a, Art 3.5; reference 5: FAO 1990b).
- Although pesticide formulations in WHO class II are less acutely hazardous than those in class I, precautionary methods proven effective under field conditions in developing countries are required. Therefore, pesticide formulations in WHO class II should only be provided if it can be demonstrated that users adhere to the necessary precautionary measures (9). (FAO 1994, Art 3.2; reference 9: FAO 1992).

The FAO has further recommended that

- integrated pest management (IPM) should be promoted by governments and other stakeholders and that even where a control scheme is in operation, the pesticide industry should cooperate in the periodic reassessment of the pesticides which are marketed.
- Industry halt sales and recall products when handling or use pose an unacceptable risk under any use directions or restrictions (FAO 2002a, Art 5.2).

On the distribution and use of acutely toxic pesticides in developing countries, the positions of the FAO, the World Health Organisation (WHO) and of the Organisation for Economic Cooperation and Development (OECD) largely correspond to each other:

'*Pesticides belonging to WHO Acute Toxicity Class Ia or Ib, respectively, should not be used in developing countries, and if possible pesticides of class II should also be avoided'* (Plestina 1984).

'*Extremely and highly hazardous pesticides of the WHO Class Ia and Ib (...) and compounds which are highly persistent in the environment should not be provided. Exceptions could only be considered if all three of the following criteria are met: a) there are urgent reasons to use these pesticides; b) there are no safer*

alternatives; and c) their safe and controlled application can be guaranteed. Pesticides of Class Ia, Ib and the more toxic range of class II, are generally considered to be unsuitable for use by smalls–cale farmers' (OECD 1995).

11. 1. 2 International Programme on Chemical Safety

The International Programme on Chemical Safety (IPCS) has pointed out that fatalities have resulted from inappropriate behaviour during the use of paraquat, such as using a leaking sprayer which may lead to severe skin lesions and absorption. Further, that damage to skin or eyes and nosebleed through the irritant action of paraquat illustrate the need for strict personal hygiene and rigourous adherence to safe handling procedures (IPCS 1991). It recommended:

- that the summary of the safety guide on paraquat should be easily available to users and to all health workers concerned with the issue;
- the safety guide be displayed on equipment at, or near, entrances to areas where there is potential exposure to paraquat, and be translated into the appropriate language (IPCS 1991, point 6).

Regarding the distribution and use of paraquat, it recommended that where practical and reasonable, the availability and use of the 20% liquid product should be limited to bona fide agriculturalists, horticulturalists, and professional users, who work with trained personnel, properly maintained equipment, and adequate supervision (IPCS 1991, section 3.2).

Recommendations on personal protection during the use of paraquat are:

- Avoid all contact with skin, eyes, nose, and mouth, when handling concentrated paraquat.
- Wear PVC, neoprene or butylrubber gloves (preferably gauntlet form), neoprene apron, rubber boots and faceshield.
- Wear a faceshield when handling and applying the diluted formulation. (...)
- Paraquat should not be sprayed with inadequate dilution, e.g., by handheld, ultralowvolume application.
- Paraquat should not be used by people suffering from dermatitis or by people with wounds, notably on the hands, until these have healed (IPCS 1991, section 4.1).

It was also specified that protective clothing should be impervious to liquids (IPCS 1991, section 6).

11. 1. 3 Intergovernmental Forum on Chemical Safety (IFCS)

The fourth Intergovernmental Forum on Chemical Safety (Forum IV, 2003) pointed out that certain aspects of the problem of pesticide poisoning will be addressed by the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade.

The IFCS Forum IV in Thailand 2003 requested that the Forum Standing Committee provide information on the extent of acutely toxic pesticides, and provide guidance for sound risk management and reduction, including options for phasing out where appropriate (IFCS 2003a). But at the international level in general, only broad requirements are referred to in laws. Requirements are met on a voluntary basis by the responsible users, producers or transporters (IPCS 2004).

Forum IV recommended that Conventions and Guidelines of the International Labour Office regarding occupational health and chemical safety be implemented such as Convention 169 on the work conditions of indigenous populations to prevent the use of specially dangerous pesticides (IFCS 2003a). The Forum made several recommendations to governments for regulatory actions aimed at reducing the risks from acutely toxic pesticides:

- prohibit or restrict availability (including the use of import and/or export controls as desirable) and use of acutely toxic pesticides (such as formulations classified by WHO) as Extremely Hazardous (Class Ia) and Highly Hazardous (Class Ib) and/or those pesticides associated with frequent and severe poisoning incidents;
- substitute acutely toxic pesticides with reduced risk pesticides and

nonchemical control measures;

- encourage industry to extend product stewardship and to voluntarily withdraw acutely toxic pesticides when poisoning incidents occur. (IFCS 2003, pg. 11).

As paraquat has been associated with 'frequent and severe poisoning incidents', urgent action is needed to implement the necessary measures to eliminate or minimise the occurrence of poisonings. This is required to prevent harm.

11. 1. 4 Rotterdam Convention

The Rotterdam Convention on the Prior Informed Consent (PIC) Procedure for Certain Hazardous Chemicals and Pesticides in International Trade regulates the information flow concerning the import and export of hazardous pesticides. Annex III of the convention list those chemicals regulated by the convention.

Banned or severely restricted chemicals (incl.

Table 3: Countries where paraquat is not authorized or has been banned

Country	Regulation, (year), legal documents or source	Comment
Bosnia & Herzegovina	Use banned. (2009) Decision on prohibition of the registration, import and placing on the market plant protection products containing certain active substances (official Gazette" of Bill No 47/09) in Bosnia and Herzegovina	
Cambodia	Use banned. (2003) MAFF, List of Pesticide Banned for use, annex 1 to Prokas of 15 December 2003	
European Union (27 Member States: Austria, Belgium, Bulgaria, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, United Kingdom	Use banned. (2010) Commission Regulation (EU) No 15/2010 of 7 January 2010 amending Annex I to Regulation (EC) No 689/2008 of the European Parliament and of the Council concerning the export and import of dangerous chemicals.	Several EU Member States prohibited the use of paraquat before: Austria (1993), Denmark (1995), Finland (1986), Slovenia (1997), Sweden (1983), Hungary (1990), Lithuania, Latvia, Estonia
Syria	Use banned. (2005) Ministry of State for Environental Affairs off the Syrian Arab Republic (MSEA)	Personal communication by Ms. Afraa Nouh to F. Meienberg in September 2005
Kuwait	Use banned. (1985)	
Sri Lanka	Use banned. (01.01.2011). UNEP (2010)	Reason for ban was the high number of suicides.
Ivory Coast	Use banned. (2004)	
Norway	No product authorization. (1996)	
Switzerland	Use banned. (1989) Swiss government.	

pesticides) and severely hazardous pesticide formulations can be listed in Annex III, if at least two parties[17] of different regions undertook regulatory actions and a certain procedure (notification of legal actions, approval by a Chemical Review Committee, vote by UN parties) has been accomplished (PIC Secretariat 2008). If intentional misuse (incl. suicides) is the sole or main reason for the national regulatory action, the proposal will be rejected.

In the seven years since the convention entered into force, one agricultural pesticide (lindane/ gamma–HCH) was additionally[18] included in Annex III. Four more are recommended for inclusion (endosulfan, alachlor, aldicarb, azinphos-methyl).

Burkina Faso recently proposed to add Gramoxone Super containing 200 g/L paraquat to the Annex III of the PIC Convention (PIC Secretariat 2010). In 2010, Sweden, Uruguay and Sri Lanka notified their regulatory actions reg. paraquat. Other countries also banned paraquat (see Table 3).

11. 1. 5 PIC Procedure of the European Union

The Rotterdam Convention (also called PIC Convention) allows parties the right to take action that is more stringently protective of human health and the environment than that called for in the Convention. The European Union recognized that ‚*It is necessary and appropriate, in order not to weaken the level of protection afforded to the environment and to the general public of importing countries (...) to go further than the provisions of the Convention in certain respects*' (EC 2008b pg. 2).

Exports of dangerous chemicals that are banned or severely restricted within the EU (listed in Annex I of the regulation) are subject to a export notification procedure. Exporters and importers are obliged to provide information concerning the quantities of chemicals in international trade. Basically, exporters have to notify the designated national authority (DNA) of the Member State in which he is established before the export of the chemical takes place. The DNA communicates the notifications with the European Commission, which seeks consent from the importing nation. Paraquat, paraquat dichloride and Paraquat bis(methylsulfate) are already included in the EU PIC procedure (EC 2010, EC 2011). The Commission maintains a database online[19], where all trade with paraquat can be observed.

12. Labels and standards prohibiting paraquat

Many companies processing food and fiber and also large retailers want to process raw-materials and sell products from more sustainable and equitable ('fair') production systems. And the market for fair-trade products is expanding. Many consumers in the industrialised countries do not want to knowingly destroy the environment and peoples' livelihoods. In response to these demands, during the last 15 years many organisations have initiated labels and certification systems. Today a large number of producers fulfill the requirements of these labels. All the main initiatives have incorporated specific requirements for pesticides. The use of paraquat has been prohibited on a voluntary basis by major certification organisations (Fairtrade Labelling Organisations, Forest Stewardship Council, Rainforest Alliance, UTZ), companies (e.g. Chiquita, Dole) and international initiatives (Common Code for the Coffee Community, RSPO) (see table 4). A large number of producers certified under these voluntary standards have demonstrated that effective and economic alternatives to paraquat exist. In most cases, the reason for the ban is the rating of paraquat as 'Dirty Dozen' pesticide (PANNA 1995) and the EU ban.

17 State or regional economic integration organisation that has consented to be bound by this Convention and for which the Convention is in force

18 As the convention entered into force Annex III was already established and contained a number of pesticides.

19 http://edexim.jrc.ec.europa.eu/

Table 4: Standards/Labels in which paraquat is banned or phased out

Standard/ Label	Description	Ban/ phase out Reason
Forest Stewardship Council (FSC)	The Forest Stewardship Council (FSC) is an international network for promoting a more sustainable management of timber plantations and forests. Over the past ten years, 135.07 million ha in more than 80 countries have been certified on the basis of FSC standards (Feb. 2011), while several thousand products made from FSC certified wood carry the FSC label. www.fsc.org	Ban. High acute toxicity. Potential to bioaccumulate. Persistence in soil.
Rainforest Alliance	The mission of the sustainable agriculture programme is to integrate productive agriculture, biodiversity conservation and human development. The area certified by the Rainforest Alliance in Latin America comprises 129.097 hectares. The majority are banana planta-tions (including all Chiquita plantations) with 46% of the total area, followed by coffee (42%), cacao (7%) and citrus (5%). (Figures from 2005.) www.rainforest-alliance.org	Ban. Paraquat is banned in the European Union and a PAN Dirty Dozen Pesticide.
UTZ Certified	UTZ CERTIFIED is coffee certification programs worldwide, and is expanding to become a multi-commodity program including cacoa, tea and palm oil. Their vision is to achieve sustainable agricultural supply chains, that meet the growing needs and expectations of farmers, the food industry and consumers alike. In 2008 approximately 77.000 coffee farmers in 19 countries were UTZ certified. www.utzcertified.org	Ban. Paraquat is banned in the European Union and a PAN Dirty Dozen Pesticide.
Fairtrade Labelling Organisations (FLO)	FLO is the organisation that sets worldwide standards for fair trade and carries out certification. FLO fair trade standards exist for coffee, tea, cocoa, sugar, honey, banana, fresh fruit and vegetables, dried fruit, fruit juice, rice, wine, nuts and oilseed, cut flowers, ornamental plants, cotton and footballs. FLO is working with 389 certified producer organisations, representing almost 500 first level producer structures, and over 800'000 families of farmers and workers, from over 48 countries in Africa, Asia and Latin America (status in May 2004) www.fairtrade.net	Ban. Paraquat is a PAN Dirty Dozen Pesticide.
Common Code for the Coffee Community (4C)	The Common Code for the Coffee Community is a joint initiative of coffee producers, trade and industry (incl. Nestlé, Kraft Foods, Sara Lee, Aldi, Lidl and others), trade unions and social or environmental NGOs. Its objective is to establish a global code of conduct aiming at social, environmental and economic sustainability in the production, postharvest processing and trading of mainstream green coffee. www.4c-coffeeassociation.org/	Phase out. Documented major poisonings.
ProTerra	ProTerra is a standard for any commodity crop producer, sugar cane, maize, soya, tobacco, palm, coffee etc. – the ProTerra® Standard emerged from the Basel Criteria, a document developed cooperatively by the retailer COOP-Switzerland, the World Wildlife Fund, and other industry and public interest groups. Approximately 50% of all imported soybeans in Switzerland are ProTerra certified. http://www.cert-id.eu/Certification-Programmes/ProTerra.aspx	Ban. Paraquat is a PAN Dirty Dozen Pesticide.
RSPO	In response to the urgent and pressing global call for sustainably produced palm oil, the Roundtable on Sustainable Palm Oil (RSPO) was formed in 2004 with the objective promoting the growth and use of sustainable oil palm products through credible global standards and engagement of stakeholders. www.rspo.org/	Phase out. High Toxicity.

13. Food companies prohibiting paraquat

Early 2011, the large UK retailer Co-op (The Co-operative Group, 5.4 percent market share) announced that use of paraquat will be phased out in their supply chain[20] (see Figure 10). By demanding that all food suppliers adopt the same Code of Practice, which includes pesticides bans, the Co-op uses its purchasing power as a retailer to raise standards beyond the boundaries of its own agricultural operations. And Co-op is not the only company prohibiting paraquat. A survey by the Berne Declaration and IUF[21] (BD/IUF 2009) showed that many leading companies, such as Chiquita, Dole and Nestea have already phased out the use of paraquat in their production/supply chain.

'*Dole's implementation of this phase-out program responds to developing marketplace conditions in Europe and elsewhere regarding the use of this herbicide, while also balancing needed compliance with the local regulatory requirements.*' stated David DeLorenzo, President and Chief Executive Officer of Dole Food Company, Inc.[22]

George Jaksch (Director of Corporate Responsibility and Public Affairs at Chiquita) said: '*Under its Better Banana Project, Chiquita is working to improve the safety of its workers and the protection of the environment on its plantations. Because we are serious in pursuing these objectives we have banned products like paraquat from our plantations.*'[23]

Unilever, the world's largest tea producer, aims to have a paraquat-free supply chain and is taking major steps to make this a reality (BD/IUF 2009).

20 http://www.co-operative.coop/corporate/Press/Press-releases/Headline-news/Join-The-Revolution/

21 International Union of Food, Agricultural, Hotel, Restaurant, Catering, Tobacco and Allied Workers' Associations (IUF)

22 http://investors.dole.com/phoenix.zhtml?c=231558&p=irol-newsArticle_pf&ID=1362807&highlight

23 Website of Berne Declaration: http://www.paraquat.ch/witnesses.cfm?p=3

14. Pesticide industry initiatives

The agrochemical industry has made efforts to promote improved pesticide use practises in developing countries. However, it was concluded that ensuring greater responsibility in the use of pesticides was an immense task, and that significant progress could be made only if academia, aid donors, government, industry, international organisations and NGOs pooled their resources, and the process was institutionalised (Vlahodimos 1999).

In 1991 the pesticide industry carried out 'Safe Use Pilot Projects' in Guatemala, Kenya and Thailand to educate and train farmers, retailers and doctors, protect people and the environment, prevent and treat contamination, and recycle or dispose of empty containers (Croplife International 1998). Objectives of the projects were to achieve a significant and measurable improvement in meeting the latest international safety standards. Also that other organisations should be stimulated to develop similar initiatives in other countries (Croplife

Figure 10: Announcement of the UK based Co-operative to prohibit its supplier the use of paraquat http://www.co-operative.coop/join-the-revolution/our-plan/Environment/Pesticides/

International 1998).

The industry claims that 956,000 farmers, 3,875 retailers, 5,000 extension staff or trainers and 3,350 doctors or medical staff were reached. The task is too large to be met by industry alone. In countries where the need for improvement is greatest, the possibilities for providing modern technology are limited. Ultimately the success of this and similar programmes appears to depend largely on the ability of the industry to integrate workers and public health scientists into the design, implementation and evaluation stages of the project (Fenske & Simcox 2000).

Similar programmes were carried out in China, India, Mexico, Philippines and Malaysia (Syngenta 2003). But it appears that the proportion of farmers reached by this programme was very low.

A large–scale project in India, Mexico and Zimbabwe studied how less hazardous ways of using pesticides could be achieved in developing countries. Farmers' knowledge, attitudes, and practices regarding personal safety during spraying, storage of pesticides, disposal of empty containers, identification of pests and product selection were assessed and the impact of communication campaigns was evaluated. Some improvements were noted. However, a large number of farmers did not improve practices even though they were aware of the health risks. Reasons for this included the need for more time, aversion to taking financial risks from change in practices (due to poverty) and external factors (weather, climate and economic situation). It was found that communication campaigns needed to be carried out continually so that changes in practices persisted, and that the family of farmers and community needed to be included in programmes to have an impact. Many if not most farmers appeared to give low priority to safety and did not adopt the necessary precautions to reduce health risks, indicating that there were limits to the extent to which changes will be adopted within a generation. Given the finding that not everyone can adopt relatively simple changes in behaviour while recognising the need for educating farmers in practices that reduce the risks it was concluded that besides subsidising suitable protective clothing, manufacturers who could not guarantee that pesticides in WHO class Ia and Ib can be used safely should withdraw these products from the market (Atkin & Leisinger 2000).

Training related to pesticides needs to be set in the broader context of sustainable agriculture and IPM, in a manner which does not engender a false sense of security that toxic chemicals are 'safe'. The message should remain focused on the hazards of pesticides namely that pesticides are the problem, not the farmer (Dinham 1995). It has been argued that knowledge was inadequately linked with structural constraints on behaviour in the industry's 'Safe Use' campaign. Industrial hygiene approaches could be applied to reducing pesticide hazards (Murray & Taylor 2000).

While educational programmes in some regions may have raised working standards, they must reach other major user groups with a high exposure and need to be evaluated by an independent agent if the outcome is to be sustained (Hurst 1999). In Indonesia a health module used in IPM farmer field schools aimed at the prevention of pesticide poisoning. This was defined as the exposure through low use (or none) of only the least hazardous pesticides (WHO class III or class U). It was based on the assessment that training farmers could not guarantee reduced exposure in the local setting (Murphy et al. 1999). Clearly the best means of protection from hazardous pesticides is not to use pesticides or to use nontoxic pesticides (Watterson 1988).

Industry has repeatedly asserted that paraquat is safe to users under 'normal conditions' (Syngenta 2005; Kurniawan 1996). But under the prevailing working conditions in developing countries, paraquat poisoning poses a severe health problem in many countries and there is a need for independent risk assessment (Wesseling et al. 2005; Hurst 1999). Since workers can absorb spray by breathing through the mouth, airborne paraquat spray (or drift) presents a high risk. Application methods producing fine droplets must not be used to spray paraquat (Pasi 1978).

The availability of products responsible for poisonings needs to be restricted (IFCS 2003). A number of companies adhering to a responsible care programme for the chemical industry have pledged to limit the marketing of products or cease production, regardless of economic interests, if the results of a risk assessment call for such limitation or cessation

as a precautionary measure to protect human health and the environment (CSC 2005). The global 'Responsible Care' programme asserts that companies evaluate their products in a rigourous manner to protect public health and the environment (ACC 2005). This has obviously not been done for paraquat.

15. Alternatives to paraquat

15. 1 Alternatives to the use of paraquat

The paper 'Weed management for developing countries' (FAO 2003) gives an overview of current practices for weed management and alternatives to the use of herbicides.

In the chapter by Paolo Bàrberi on preventive and cultural methods for weed management, Bàrberi states that in many agricultural systems around the world, competition from weeds is still one of the major factors reducing crop yield and farmers' income. At worldwide level, the limited success in weed control is probably the result of an oversimplification in tackling the problem. Too much emphasis has been given to the development of weed control, especially synthetic herbicides, as the ultimate solution to all weed problems, while the importance of integrating different tactics (e.g. preventive, cultural, mechanical, and chemical methods) in a weed management strategy based on the crop system has long been neglected (FAO 2003). Agricultural practice has demonstrated that the philosophy of integra ted management used in insect control needs to be similarly adopted in weed control (ibid.).

Integrated weed management is based on the knowledge of the biological and ecological characteristics of weeds. This knowledge can increase understanding of how weeds can be regulated by cultural practices. A long-term effective weed management strategy is based on the practical application of the concept in ecology of 'maximum diversification of disturbance', which means diversifying crops and cultural practices in a given agroecosystem as far as possible (FAO 2003).

The highest diversification of the cropping system (i.e. crop sequence and associated cultural practices) based on agroecological principles is the key to effective long-term weed management in any situation. In this respect, the systematic inclusion of preventive and cultural methods for weed management must always be pursued (FAO 2003). An overview of cultural methods of weed control is presented in table 5.

In humid climates, weeds are more of a problem than in the temperate zones. Parasitic weeds (Striga, Orobranche or Cuscuta) in the tropics can damage the crop. Weeds can generally be controlled effectively through an appropriate crop rotation, trap crops and good soil management (Neubert & Knirsch 1996). Maize and other crops have been found to display tolerance (lower yield loss) and even resistance towards Striga species and cultivating tolerant and resistant crops is a viable pesticidefree option of controlling Striga (Pingali & Gerpacio 1998).

In minimum tillage systems, paraquat is used to kill vegetation before direct seeding of the crop (Bromilow 2003). But large areas are cultivated by minimum tillage without the use of paraquat (BLW 2001). Mechanical removal of cover crops was shown to be more economical than the use of paraquat in the US (Ashford & Reeves 2001). Furthermore at least 25 weeds are reported to be resistant to paraquat (WSC 2009).

Alternatives to herbicides are commonly used in organic farming. Organic agriculture is practised in 160 countries and 37.2 million hectares of agricultural land are managed organically by 1.8 million farmers[24], with no use of synthetic herbicides at all.

Alternatives are also part of Integrated Pest Management (IPM), which reduces the use of pesticides as much as possible. The IPM Danida project in Thailand has a very clear standpoint regarding the use of paraquat in IPM: '*The most dangerous chemicals, including all class Ia and Ib pesticides and paraquat should be banned immediately. They have no place in IPM because less risky alternatives are available*' (IPM DANIDA 2004).

According to IOBC (International Organisation for Biological and Integrated Control of Noxious Animals and Plants an organisation that aims at promoting the development of biological control weed management should be achieved, as far as possible, by non-chemical

24 http://www.organic-world.net

Table 5: Classification of cultural practices potentially applicable in an integrated weed management system, based on their prevailing effect (FAO 2003)

Cultural practice	Category	Prevailing effect	Example
Crop rotation	Preventive method	Reduction of weed emergence	Alternation between cereal/ broadleave; summer/winter crops
Cover crops (used as green manures or mulches)	Preventive method	Reduction of weed emergence	Cover crop grown in-between two cash crops
Primary tillage	Preventive method	Reduction of weed emergence	Deep ploughing, alternation between ploughing and reduced tillage
Seed bed preparation	Preventive method	Reduction of weed emergence	False (stale)-seed bed technique
Soil solarization	Preventive method	Reduction of weed emergence	Use of black or transparent films (in glasshouse or field)
Irrigation and drainage system	Preventive method	Reduction of weed emergence	Irrigation placement (micro/trickle-irrigation), clearance of vegetation growing along ditches
Crop residue management	Preventive method	Reduction of weed emergence	Stubble cultivation
Sowing/planting time and spatial arrangement	Cultural method	Improvement of crop competitive ability	Use of transplants, higher seeding rate, lower inter-row distance, anticipation or delay of sowing/ transplant date
Crop genotype choice	Cultural method	Improvement of crop competitive ability	Use of varieties characterised by quick emergence, high
Cover crops (used as living mulches)	Cultural method	Improvement of crop (canopy) competitive ability	Legume cover crop sown in the inter-row of a row crop
Intercropping	Cultural method	Reduction of weed emergence, improvement of crop competitive ability	Intercropped crops
Fertilization	Cultural method	Reduction of weed emergence, improvement of crop competitive ability	Use of slow nutrient-releasing organic fertilizers and amendments, fertilizer placement, anticipation or delay of pre-sowing or top-dressing N fertilization
Mechanical cultivation	Curative method	Killing of existing vegetation, reduction of weed emergence	Post-emergence harrowing or hoeing, ridging
Herbicide application	Curative method	Killing of existing vegetation, reduction of weed emergence	Pre- or post-emergence spraying
Thermal weed control Curative method	Curative method	Killing of existing vege tation,reduction of weed emergence	Pre-emergence or localized post-emergence flame-weeding
Biological weed control Curative method	Curative method	Killing of existing vegetation	Use of (weed) species-specific pathogens reduc tion of weed emergence

methods. Nonselective pesticides with a long persistence (slow degradation), high volatility, and/or human-toxicity high toxicity to mammals/humans [high acute mammalian toxicity] are prohibited. The ban on Paraquat is explicitly listed in the IOBC Crop Specific Technical Guidelines III for olives, grapes and soft fruits (IOBC 2004)[25].

In Indonesia, from 1993 to 1998, IPM lowered the health costs associated with pesticide poisoning by nearly 2%. For rice farming, total health costs related to pesticide poisoning dropped by about 5% and the efficiency of rice production improved in the same period. (Resudarmo 2000).

More proof of viable alternatives to paraquat is provided by the timber from millions of hectares which is certified by the Forest Stewardship Council and from crops certified by the Rainforest Alliance (banana, coffee, citrus, cacao), and the Fairtrade Labelling Organisations (coffee, tea, cocoa, sugar, honey, banana, fruit, vegetables, rice, wine, nuts, oilseed, flowers and cotton) (see Table 5).

A closer look at alternatives to herbicides in general, and paraquat in particular, requires that the situation be analysed separately for each crop. A comprehensive overview is not possible within this report, but coffee and banana are presented below as examples for the discussion.

Coffee

A survey covering 34 farms and plantations in Latin America showed that 59% relied only on mechanical weeding (machete, hoe or motor scythe), 41% relied on mechanical weeding and herbicides, 12% were using paraquat. Another 12% of respondents said they used paraquat before but not anymore (Menet 2002).

The recommendations for agrochemicals of the Common Code of the Coffee Community (4C) state that in well established plantations, when working with mulch as soil cover, there is normally no need for herbicides (Jansen 2005). The situation is different during establishment of plantations, especially for sungrown coffee or while shade trees are not fully developed. At this stage the crop is more sensitive to competing weeds and cannot suppress them. The growth of weeds, says the Code, should normally be controlled with cover crops as far as possible. Hand weeding should be employed as far as labour is available and the costs are reasonable. This strategy may be complemented by a herbicide of relatively low acute toxicity (in WHO class III or U) and with a low potential of leaching to avoid groundwater contamination.

To reduce the use of pesticides, specific IPM recommendations were developed for farmers in Tanzania on how to manage their most important problems with coffee (Jansen 2005). They include shade management, intercropping with bananas, organic manure and mulching, irrigation techniques and weeding when ground cover is over 50%, i.e. about 45 times per year. Nishimoto (1994) has pointed out that the most promising practices for an appropriate low input or sustainable scheme of weed control in coffee plantations are the use of shade trees, leguminous cover crops and mulching. In South and Central America cover crops are often legumes, which have an additional benefit from nitrogen fixation (PAN UK 1998).

One of the biggest coffee traders worldwide, Volcafe, has stopped using a paraquat on its plantations. A company representative told the Berne Declaration: 'We are of the opinion that paraquat is not a suitable product to control weeds. In particular its toxicity causing high risks for users, but also economical reasons speak against the product. There are alternatives today which are cheaper and more secure' (Volcafe 2003).

Bananas

Weeds are a problem in bananas and plantains because they compete for water, nutrients and light. Practices commonly used for weed management are described by FAO as:

- Mechanical weed control: Slashing the weeds 34 times a year and leaving the weed mulch on the surface will help avoid soil erosion, delay fresh weed growth (but not eliminate weeds) and allow access. Slashing has to be done with care, or else banana stems and suckers will be damaged.
- Cultural weed control (healthy

25 IOBC (2004): Integrated Production Principles of IOBC. International Organisation for Biological and Integrated Control of Noxious Animals and Plants (IOBC) http://www.iobc-wprs.org/ip_ipm/index.html

planting material and close spacing of the crop, cover plants and mulch). Cover plants can be used to suppress weed growth and have been widely recommended. Small farmers are likely to want cover plants that can be utilised. Good results have been achieved with watermelons in West Africa, cowpeas in India or with sweet potatoes. Kotoky and Bhattacharyya (quoted by Terry (1994) showed that the bunch weight and yield could be significantly increased when mulch was applied (36 tons of rice straw per hectare).

- Chemical weed control: Economics of herbicide use varies around the world. Herbicide use is often impractical or inappropriate, especially in poorer developing nations. Using glyphosate is an option but should not be perceived as a panacea for all weed problems in bananas. Herbicides have the capacity to solve problems as well as to create them. (Terry 1994)

Chiquita made some substantial achievements in reducing herbicide use during the last few years. Under the Better Banana Project of the Rainforest Alliance, the use of paraquat was phased out in all their plantations. Chiquita officials stated that production has not suffered and that the programme achieved cost savings by getting so many farms involved in common practices, including a reduction in herbicide use by as much as 80 percent. This reduction has been possible trough Integrated Crop Management practices such as shade growing, mulching and ground cover with cover crops such as Geophila repens. Chiquita found that manual weed control (by machete) is efficient, but linked with increased costs. Where difficulties occurred in establishing a ground cover, the weed species were either aggressive or high rainfall favoured the rapid growth of weeds. The herbicide used most often was glyphosate (Jaksch 2002[26]). Chemical control should be reduced to the minimum, and preferably replaced entirely by cultural methods.

In banana plantations certified by Fairtrade Labelling Organisations International (FLO) the use of herbicides is banned. In one such plantation in Colombia, weed management is carried out with a machete about every 40 days (Mercado 2002). An organic banana producer in the Philippines controls weeds every 34 weeks, especially when plants are newly planted. Plant residues are left to decompose around the stem, without coming into contact with the body of the standing plant. Additionally weeds are suppressed by mulching with cut grass and leaves (Astorga 1998).

26 Personal communication with George Jaksch (Chiquita) to Berne Declaration, July 2002

16. References

A

[1] Abdel Rasoul GM, Abou Salem ME, Mechael AA, Hendy [1] OM, Rohlman DS & Ismail AA (2008): Effects of occupational pesticide exposure on children applying pesticides. Neurotoxicology (5):833-838.

[2] ACC (2005): Responsible Care: managing product safety. American Chemistry Council (ACC).

[3] Alberdi JL, Sáenz ME, Di Marzio WD, and Tortorelli MC (1996): Comparative acute toxicity of two herbicides, paraquat and glyphosate, to Daphnia magna and D. spinulat. Bulletin of Environmental Contamination and Toxicology 57, 229-235.

[4] Ambrus A, Hamilton DJ, Kuiper HA, and Racke KD, Significance of impurities in the safety evaluation of crop protection products, Pure and Applied Chemistry 75(7), 937-973, 2003

[5] Ames R, Howd R, and Doherty L (1993): Community exposure to paraquat drift. Archives of Environmental Health 48(1):47-52.

[6] Andersohn C (2002): Phytotoxische Wirkungen von gelöster organischer Substanz (DOM) mit Paraquat, 2,4-D und Naphthalin (PhD thesis), Berlin: Technische Universität

[7] Angehrn B (1996): Plant protection agents in developing country agriculture: Empirical evidence and methodological aspects of productivity and user safety (PhD thesis), Zurich: ETH

[8] Arroyave ME (1993): Pulmonary obstructive disease in a population using paraquat in Colombia. In: Impact of pesticide use on health in developing countries. International Development Research Centre. Canada http://collections.infocollections.org/ukedu/en/d/Jid22ie/3.9.html assessed: March 17th 2011.

[9] Ascherio A, Chen H, Weisskopf MG, O'Reilly E, McCullough ML, Calle EE, Schwarzschild ME & Thun MJ (2006): Pesticide exposure and risk for Parkinson's disease. Annals of Neurology 60(2):197 - 203.

[10] Ashford D & Reeves W (2001): Rolling and crimping: scientists study alternative cover crop kill method, Highlights Online 48(3).

[11] Astorga Y (1998): The environmental impact of the banana industry: a case study of Costa Rica.

[12] ASU (2001): Paraquat. Agriculture Siam Universal Co. Ltd., (ASU). Thailand 2001

[13] Athanaselis S, Quammaz S, Alevisopoulos G & Koutselinis A (1983): Percutaneous paraquat intoxitation, Journal of Toxicology: Cutaneous and Ocular Toxicology 2:3-5.

[14] Atkin J & Leisinger K (eds.) Safe and effective use of crop protection products in developing countries, Oxford: CABI Publishing 2000

[15] ATS (American Thoracic Society), Respiratory hazards in agriculture (chapter 4), American Journal of Respi-ratory and Critical Care Medicine 158 (5 Pt 2), S1-S76, 1998

[16] ATS and ERS, (American Thoracic Society and European Respiratory Society), Idiopathic pulmonary fibrosis: diagnosis and treatment - International consensus statement, American Journal of Respiratory and Critical Care Medicine 161(2), 646-664, 2000

[17] Atuniassi UR, and Gandolfo MA, Periodic inspection of crop sprayers: results according to age of sprayer, Journal of Environmental Science and Health Part B 40(1), 195-200, 2005

B

[18] Bade BL, Is there a doctor in the field? Underlying conditions affecting access to health care for California farmworkers and their families, CPRC report, California: University of California 1999

[19] Ballantyne B, Marrs TC & Turner P (eds.) (1995): General & applied toxicology (abridged edition), 474-476, Basingstoke, England: Macmillan.

[20] Ballard TJ & Calvert GM (2001): Surveillance of acute occupational pesticide-related illness and injury: the US experience. Annali dell' Istituto Superiore di Sanita 37(2), 175-179.

[21] Balme KH, Roberts JC, Glasstone M, Curling L, Rother HA, London L, Zar H & Mann MD (2010): Pesticide poisonings at a tertiary children's hospital in South Africa: an increasing problem.Clinicical Toxicology 48(9):928-34.

[22] Bartlett RM, Holden JE, R. Nickles J, Murali D, Barbee DL, Barnhart TE, Christian BT & DeJesus O (2009): Paraquat is excluded by the blood brain barrier in rhesus macaque: An in vivo pet stud. Brain Research (1259): 74-79.

[23] Baselt RC, and Cravey RH, Paraquat, In: Disposition of toxic drugs and chemicals in man, 3rd ed., 637-640, Chicago: Year Book Medical Publishers 1989 (quoted by Garnier (1995))

[24] Baselt RC, Paraquat, In: Biological monitoring methods for industrial chemicals, 2nd ed., 240-243, Littleton, Ma.: PSG Publishing 1988 (quoted by Garnier (1995))

[25] Bataller R, Bragulat E, Nogue S, Gorbig MN, Bruguera M & Rodes J (2000): Prolonged cholestasis after acute paraquat poisoning through skin absorption, American Journal of Gastroenterology 95(5): 1340-1343.

[26] Bateman DN (2008): New formulation of paraquat: a step forward but in the wrong direction. PloS Medicine 5(2): e58 (http://dx.doi.org/10.1371/journal.pmed.0050058)

[27] BD/IUF (2009): Goodbye Paraquat. Palm Oil, Banana and Tea Producers Saying No to Hazardous Pesticide. International Union of Food, Agricultural, Hotel, Restaurant, Catering, Tobacco and Allied Workers' Associations (IUF)

[28] and the Berne Declaration

[29] Beligaswatte AM, Kularatne SA, Seneviratne AB, Wijenayake MS, Kularatne WK & Pathirage LM (2008): An outbreak of fatal pneumonitis caused by contamination of illicit alcohol with paraquat. Clinical Toxicology 46 (8): 768-770.

[30] Berry C, La Vecchia C & Nicotera P (2010): Paraquat and Parkinson's disease. Cell Death and Differentiation 17:1115–1125.

[31] Bertsias GK, Katonis P, Tzanakakis G & Tsatsakis AM (2004): Review of clinical and toxicological features of acute pesticide poisonings in Crete (Greece) during the period 1991-2001, Medical Science Monitor 10(11), CR 622- 627.

[32] Betz H-G, Das Verhalten von Paraquat in einem ausgedehnten Entwässerungssystem: Ein Beitrag zum Problem der chemischen Grabenentkrautung (PhD thesis), Kiel: Christian-Albrechts-Universität 1975

[33] BfR (2011): Health assessment of ethephon residues in bell peppers. BfR Opinion Nr. 001/2011, 19 January 2011. Bundesinstitut für Risikobewertung BfR)

[34] Binns CW (1976): A deadly cure for lice - a case of paraquat poisoning. Papua New Guinea Medical Journal 19(2):105-7.

[35] Bismuth C, and Hall A, Pulmonary sequelae in poisong survivors, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 349-355, New York 1995

[36] Bismuth C, Ganier R, Dally S & Fourniwer PE (1982): Prognosis and Treatment of Paraquat Poisoning: A Review of 28 Cases. Clinical Toxicology 19(5): 461–474.

[37] Bismuth C, Hall A, and Wong A, Paraquat ingestion exposure: symptomatology and risk, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 195-210, New York: Marcel Dekker 1995

[38] BLW (2001): Direktsaat im Praxisversuch. Agrarforschung 8(1): 4-28. Bundesamt für Landwirtschaft (BLW)

[39] Boodram N, The fate of agro-chemicals in the land-water interface, with reference to St Lucia and the wider Caribbean, Impact and amelioration of sediment and agrochemical pollution in Carribbean coastal waters, project reports (R7668), report 4, St. Lucia, West Indies 2002

[40] Botella R, Sastre A & Castells A (1985): Contact dermatitis to paraquat, Contact Dermatitis 13(2), 123-124

[41] Bové J, Prou D,Perier C & Przedborski S (2005): Toxin-Induced Models of Parkinson's Disease. The Journal of the American Society for Experimental NeuroTherapeutics (2):484–494.

[42] Bowles JR, Suicide in western Samoa: an example of a suicide prevention program in a developing country, In: Diekstra RFW, Gulbinat W, Kienhorst I, and de Leo D (eds), Preventive strategies on suicide, 173-206, Leiden: EJ Brill 1995 (quoted by WHO (2002))

[43] Branson D & Sweeney M (1991): Pesticide personal protective clothing, Reviews of Environmental Contamination and Toxicology 122, 81-109.

[44] Bromilow (2003): Paraquat and sustainable agriculture. Pest Management Science 60:340 – 349.

[45] Brook EM (ed), Suicide and attempted suicide, Public Health Papers 58, Geneva: WHO 1974 (quoted by Wesseling et al (1993))

[46] Broughton E (2004): The Bhopal disaster and its aftermath: a review. Environmental Health: A Global Access Science Source 2005, 4:6 doi:10.1186/1476-069X-4-6

[47] Brown TP, Rumsby PC, Capleton AC, Rushton L & Levy LS (2006): Pesticides and Parkinson's Disease—Is There a Link? Environmental Health Perspectective 114:156–164.

C

[48] Cal EPA (2009): Development of Health Criteria for School Site Risk Assessment Pursuant To Health And Safety Code Section 901(g): Child-specific Reference Dose (chRD) for Paraquat. Draft Report. Integrated Risk Assessment Branch. Office of Environmental Health Hazard Assessment. California Environmental Protection Agency (Cal EPA).

[49] Calderbank A, and Farrington JA, The chemistry of paraquat and its radical, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 89-106, New York 1995

[50] Cantor A, and Young-Holt B, Pesticide-related symptoms among farm workers in rural Honduras, International Journal of Occupational and Environmental Health 8(1), 41-45, 2002

[51] Casey P, and Vale JA, Deaths from pesticide poisoning in England and Wales: 1945-1989, Human and Experimental Toxicology 13(2), 95-101, 1994

[52] Cassidy N, and Tracy JA, Morbidity and mortality following inadvertent poisoning with decanted chemicals, Irish Medical Journal 98(6), 175-178, 2005

[53] Castro-Gutiérrez N, McConnell R, Andersson K, Pacheco-Anton F, and Hogstedt C, Respiratory symptoms, spirometry and chronic occupational paraquat exposure, Scandinavian Journal of Work, Environment and Health 23(6), 421-427, 1997

[54] CDPR (1998-2003): Pesticide Illness Survey. Reports 1998-2003. California Department of Pesticide Regulation (CDPR)

[55] CDPR (2001): California Code of Regulations, California Department of Pesticide Regulation (CDPR), Division 6: Pesticides and pest control operations (last updated in July 2001)

[56] CDPR (2005): Summary of Results from the California Pesticide Illness Surveillance Program 2003. HS-1857 California Environmental Protection Agency (Cal EPA). California Department of Pesticide Regulation (CDPR).

[57] Centre d'Etude et de Développement Agricole Cambodgien (CEDAC), Pesticide use consequence in Cambodia, Phnom Penh, Cambodia 2004

[58] Chen Q, Niu Y, Zhang R, Guo H, Gao Y, Li Y & Liu R (2010): The toxic influence of paraquat on hippocampus of mice: Involvement of oxidative stress. NeuroToxicology 31:310–316.

[59] Chester G, Adam AV, Inkmann Koch A, Tuinman CP, Field evaluation of protective equipment for pesticide operators in tropical climate, La Medicina del Lavoro 81(6), 480-488, 1990

[60] Chester G, and Woollen BH, Studies of the occupational exposure of Malaysian plantation workers to paraquat, British Journal of Industrial Medicine 39(1), 23-33, 1982

[61] Chester G, Gurunathan G, Jones N, and Woollen BH Occupational exposure of Sri Lankan tea plantation workers to paraquat, Bulletin of the World Health Organisation 71(5), 625-632, 1993

[62] Chishiro T (2000): Clinical aspect of accidental poisoning with paraquat. Asian Medical Journal 43:114-20.

[63] Cone JE, Wugofski L, Balmes JR, Das R, Bowler, R, Alexeeff G & Shusterman D (1994): Persistent Respiratory Health Effects After a Metam Sodium Pesticide Spill. Chest (106):500-508. doi 10.1378/chest.106.2.500

[64] Constenla MA, Riley D, Kennedy SH, Rojas CE, Mora LE, and Stevens JE, Paraquat behaviour in Costa Rican soils and residues in coffee, Journal of Agricultural and Food Chemistry 38, 1985-, 1990

[65] Corriols M & Aragón a (2010): Child Labor and Acute Pesticide Poisoning in Nicaragua. Failure to Comply with Children's Rights. International Journal of Occupational and Environmental Health 16:193–200

[66] Cory-Slechta DA, Thiruchelvam M, Richfield EK, Barlow BK, and Brooks AI, Developmental pesticide exposures and the Parkinson's disease phenotype, Birth Defects Research part A: Clinical and Molecular Teratology 73(3), 136-139, 2005

[67] Cravey RH (1979): Poisoning by Paraquat. Clinical Toxicology 14(2):195-198.

[68] Crop Protection (M) Sdn Bhd, CSH-Paraquat (product label), Selangor, Malaysia 2004

[69] Croplife International, Safe use pilot projects, Brussels 1998. Available at

[70] CSC (2005): Responsible Care guiding principles. Ciba Specialty Chemicals (CSC). Basel, Switzerland.

D

[71] D'Souza UJ, Zain A, Raju S (2005): Genotoxic and cytotoxic effects in the bone marrow of rats exposed to a low dose of paraquat via the dermal route. Mutation Research 581 (1-2): 187-190.

[72] Daigle MS (2005): Suicide prevention through means restriction: Assessing the risk of substitution. A critical review and synthesis. Accident Analysis and Prevention 37:625–632.

[73] Daisley H & Simmons V (1999): Homicide by paraquat poisoning. Medicine science and the law 39(3):266-9.

[74] Dalvie MA, London L, and Myers JE, Respiratory health effects due to long-term low-level paraquat exposure (correspondence), American Journal of Respiratory and Critical Care Medicine 172(5), 646-647, 2005

[75] Dalvie MA, White N, Raine R, Myers JE, London L, Thompson M, and Christiani DC, Long term respiratory health effects of the herbicide, paraquat, among workers in the Western Cape, Occupational and Environmental Medicine 56(6), 391-396, 1999

[76] Damalas CA, Georgiou EB & Theodorou MG (2006): Pesticide use and safety practices among Greek tobacco farmers: A survey. International Journal of Environmental Health Research 16 (5):339 - 348.

[77] Damanakis M, Drennan DS, Fryer JD, and Holly K, The adsorption and mobility of paraquat on different soils and soil constituents, Weed Research 10, pp. 264-77, (278ff; 305ff), 1970

[78] Davanzo F, Settimi L, Faraoni L, Maiozzi P, Travaglia A, and Marcello I, [Agricultural pesticide-related poisonings in Italy: cases reported to the Poison Control Centre of Milan in 2000-2001] [Article in Italian], Epidemiologia e Prevenzione 28(6), 330-337, 2004

[79] Dawson A & Buckley N (2007): Integrating approaches to paraquat poisoning.Ceylon Medical Journal 52: 45–47

[80] Dawson AH, Eddleston M, Senarathna L, Mohamed F, Gawarammana I, Bowe SJ, Manuweera G, Buckley NA (2010): Acute Human Lethal Toxicity of Agricultural Pesticides: A Prospective Cohort Study. PLoS Medicine 7(10): e1000357.

[81] de Silva CD, and Ranasinghe J, Toxicity of four commonly used agrochemicals on Oreochromis niloticus (L.), Asian Fish Science 2, pp. 135ff, 1989 (quoted by Hall (1995))

[82] Daéid NN (1997): Suicide in Ireland 1982 to 1992. Archives of Suicide Research 3 (1): 31—42. DOI: 10.1080/13811119708258254

[83] Dick FD (2006): Parkinson's disease and pesticide exposures. British Medical Bulletin 79 and 80: 219–231. DOI: 10.1093/bmb/ldl018

[84] Dinham B (1995): Safe use or sustainable agriculture - the battle for hearts and minds. Pesticides News 28.

[85] Dinham B (2003): Growing vegetables in developing countries for local urban populations and export markets: problems confronting small-scale producer. Pest Management Science 59:575–582. DOI: 10.1002/ps.654.

[86] Dinham B (2007): Pesticide users at risk. Survey of availability of personal protective clothing when purchasing paraquat in China, Indonesia and Pakistan and failures to meet the standards of the Code of Conduct. Berne Declaration (BD), Pesticide Action Network Asia and the Pacific (PANAP), Pesticide Eco-Alternatives Center (PEAC), Gita Pertiwi, Lok Sanjh. http://www.evb.ch/cm_data/Paraquat-Code_Survey_FINAL_rev1.pdf

[87] Dinis-Oliveira RJ, Duarte JA, Sánchez-Navarro A, Remião F, Bastos ML & Carvalho F (2008): Paraquat Poisonings: Mechanisms of Lung Toxicity, Clinical Features, and Treatment. Critical Reviews in Toxicology, 38:13–71.

[88] DPI (2004): Broadacre field crops: how toxic are your herbicides on your property? Department of Primary Industries and Fisheries, Queensland Government, Brisbane, Queensland

[89] Drucker A, Cowder B, Alvarado J, Gonzalez R, and Rubio O, Economic evaluation of the occupational health impact of agrochemical use in Yucatan, Mexico, Paper presented at the conference, 25th World Congress on Occupational Safety and Health, 12-14 April, Säo Paulo, Brazil 1999 (quoted by Giuffrida et al 2001)

E

[90] EEA (2001): Late lessons from early warnings: the precautionary principle 1896–2000. Environmental issue report No 22. European Environment Agency (EEA). Copenhagen.

[91] Ellenhorn MJ, Schonwald S, Ordog G, Wasserberger J (eds), Paraquat, In: Ellenhorn's medical toxicology: diagnosis and treatment of human poisoning, 1631-1637, Baltimore, MD 1997

[92] Engel LS, Checkoway H, Keifer MC, Seixas NS, Longstreth WT Jr, Scott KC, Hudnell K, Anger WK, and Camicioli R, Parkinsonism and occupational exposure to pesticides, Occupational and Environmental Medicine 58(9), 582-589, 2001

[93] Ergonen AT, Salacin S, Ozdemir MH (2005): Pesticide use among greenhouse workers in Turkey. Journal of Clinical Forensic Medicine 12 (4) 4: 205-208.

[94] EU (1997): Council Directive 97/57/EC of 22 September 1997 establishing Annex VI to Directive 91/414/EEC concerning the placing of plant protection products on the market OJ L 265. Council of European Union (EU).

[95] EC (2003a): Commission Directive 2003/112/EC of 1 December 2003 amending Council Directive 91/414/EEC to include paraquat as an active substance (Text with EEA relevance), Official Journal L 321, 32-35. European Commission (EC).

[96] EC (2002a): Opinion of the Scientific Committee on Plants on specific questions from the Commission regarding the evaluation of paraquat in the context of Council Directive 91/414/EEC. European Commission (EC) SCP/PARAQ/00-Final.Brussels.

[97] EC (2008): Regulation (EC) No 689/2008 of the European Parliament and of the CouncIL of 17 June 2008 concerning the export and import of dangerous chemicals. Official Journal of the European Union L 204.

[98] EC (2010): Commission Regulation (EU) No 15/2010 of 7 January 2010 amending Annex I to Regulation (EC) No 689/2008 of the European Parliament and of the Council concerning the export and import of dangerous chemicals. Official Journal of the European Union L 204.

[99] EC (2011): Commission Regulation (EU) No 186/2011 of 25 February 2011 amending Annex I to Regulation (EC) No 689/2008 of the European Parliament and of the Council concerning the export and import of dangerous chemicals. Official Journal of the European Union L 53

[100] EC (2003b): Review report for the active substance paraquat. European Commission (EC). Brussel.

[101] EU (2002b): Towards a thematic strategy on the sustainable use of pesticide. European Union (EU) Brussels.

[102] EU (2007): Judgment of the Court of First Instance (Second Chamber, Extended Composition) in Case T-229/04: APPLICATION for annulment of Commission Directive 2003/112/EC of 1 December 2003 amending Council Directive 91/414/EEC to include paraquat as an active substance (OJ 2003 L 321, p. 32). Sweden vs. European Commission. 11 July 2007.

[103] EU (2008): Regulation (EC) No 1272/2008 of the European Parliament and of the Council of 16 December 2008 on classification, labelling and packaging of substances and mixtures, amending and repealing Directives 67/548/EEC and 1999/45/EC, and amending Regulation (EC) No 1907/2006. Official Journal of the European Union L 353/1.

[104] EU (2009): Regulation (EC) No 1107/2009 of the European Parliament And Of The Council of 21 October 2009 concerning the placing of plant protection products on the market and repealing Council Directives 79/117/EEC and 91/414/EEC. Official Journal of the European Union L 309.

[105] Elbaz A, Clavel J, Rathouz PJ, Moisan F, Galanaud JP, Delemott B, Alpérovitch A & Tzourio C (2009): Professional exposure to pesticides and Parkinson disease. Annals of Neurology 66 (4): 494-504.

F

[106] FAO (1986): International code of conduct on the distribution and use of pesticides. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[107] FAO (1990a): The International Code of Conduct on the Distribution and Use of Pesticides, Amended Version. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[108] FAO (1990b):Guidelines for Personal Protection when Working with Pesticides in Tropical Climates. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[109] FAO (1992): Pesticides selection and use in field projects, Field Programme Circular no. 8/92. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[110] FAO (1994a): Provisional Guidelines on Tender Procedures for the Procurement of Pesticides. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[111] FAO (2000): Paraquat (fact sheet), In: FAO, Assessing soil contamination: a reference manual, appendix 3. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[112] FAO (2002a): International Code of Conduct on the Distribution and Use of Pesticides (Revised version). Food and Agriculture Organisation of the United Nations (FAO). Rome.

[113] FAO (2002): Maximum residue limits, Rome 2004b

[114] FAO (2003): Weed management for developing countries, addendum 1, Rome (1994b; updated in 2003) Food and Agriculture Organisation of the United Nations (FAO). Rome.

[115] FAO (2003a): Paraquat dichloride. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[116] FAO (2004a): Pesticide residues in food: report of the 2003 Joint FAO/WHO Meeting of Experts. Food and Agriculture Organisation of the United Nations (FAO). Rome.

[117] FAO/WHO (2007): Report. 1st FAO/WHO Joint Meeting on Pesticide Management and 3rd Session of the FAO Panel of Experts on Pesticide Management 22–26 October 2007, Rome. Food and Agriculture Organisation (FAO) of the United Nations, World Health Organisation (WHO).

[118] Farahat FM, Fenske RA, Olson JR, Galvin K, Bonner MR, Rohlman DS, Farahat TM, Lein PJ & Anger WK (2010): Chlorpyrifos exposures in Egyptian cotton field workers. Neurotoxicology (3):297-304.

[119] Fenske RA (1988): Use of fluorescent tracers and video imaging to evaluate chemical protective during pesticide appliactions, In: Mansdorf SZ, Sager R, and Nielson AP (eds), Performance of Protective Clothing: Second Symposium, STP 989, 630-639, American Society for the Testing of Materials, Philadelphia

[120] Fenske RA, and Simcox NJ, Agricultural workers, In: Levy BS, and Wegman DH (eds), Occupational health: recognizing and preventing work-related diseases and injuries, 309-333, Philadelphia, PA 2000

[121] Fernández M, Ibáiez M, Picö Y, and Maies J, Spatial and Temporal trends of paraquat, diquat, and difenzoquat contamination in water from marshes of the Valencian community (Spain), Archives of Environmental Contamination and Toxicology 35(3), 377-384, 1998

[122] Fernando R, Harendra de Silva DG & Amarasena TSD (1990): An unusual case of fatal accidental paraquat poisoning. Forensic Science International 44 (1): 23-26.

[123] Fernando R, Harendra de Silva DG, and Amarasena TSD, An unusual case of fatal accidental paraquat poisoning, Forensic Science International 44(1), 23-26, 1990

[124] Firestone JA, Smith-Weller T, Franklin G, Swanson P, Longstreth WT & Checkoway H (2005): Pesticides and Risk of Parkinson Disease A Population-Based Case-Control Study. Archives of Neurology 62:91-95

[125] Fischer H & Kahler J (1979): Zur tödlichen Paraquatvergiftung. Fallbeschreibung. Zeitschrift für Rechtsmedizin 84:61–67

[126] Fitzgerald GR, Carmody M, Barniville G, O'Dwyer WF, Black J, and Silke B, Paraquat poisoning in agricultural workers, Journal of the Irish Medical Association 71(10), 336-342, 1978 (quoted by Garnier (1995) and by Wesseling et al (1997)

[127] Foro Emaus, Bananas for the World and the negative consequences for Costa Rica, 1998

[128] Friedrich T, Agricultural pesticide application: concepts for improvements, Rome (Food and Agriculture Orga-nisation of the United Nations), 2000

[129] Frumkin H, Toxins, In: Levy BS, and Wegman DH (eds), Occupational health: recognizing and preventing work-related diseases and injuries, 309-333, Philadelphia, PA 2000

[130] Fryer JD, Hance RJ, and Ludwig JW, Long-term persistence of paraquat in a sandy loam soil, 189-194, Weed Research 15, 1975

G

[131] Garcia AM, Benavides FG, Fletcher T, Orts E, Paternal exposure to pesticides and congenital malformations, Scandinavian Journal of Work Environment and Health 24(6), 473-480, 1998

[132] Garnier R, Chataigner D, Efthymiou ML, Moraillon I & Bramary F (1994): Paraquat poisoning by skin absorption: Report of two cases. Veterinary & Human Toxicology 36: 313–315.

[133] Garnier R, Paraquat poisoning by inhalation or skin absorption, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 211-234, New York: Marcel Dekker 1995

[134] Gear AJ, Ahrenholz DH, and Solem LD, Paraquat poisoning in a burn patient, Journal of Burn Care and Rehabilitation 22(5), 347-351, 2001 (discussion p. 346)

[135] Gunnell D, Eddleston M, Phillips MR & Konradsen F (2007a): The global distribution of fatal pesticide self-poisoning: Systematic review. BMC Public Health 7:357 http://www.biomedcentral.com/1471-2458/7/357

[136] Gunnell D, Fernando R, Hewagama M, Priyangika WDD, Konradsen F & Eddleston M (2007b): The impact of pesticide regulations on suicide in Sri Lanka. International Journal of Epidemiology 36:1235–1242 Free Access: http://ije.oxfordjournals.org/cgi/content/full/dym164v1/DC1

[137] Guo YL, Wang BJ, Lee CC, and Wang JD, Prevalence of dermatoses and skin sensitisation associated with use of pesticides in fruit farmers of southern Taiwan, Occupational and Environmental Medicine 53, 427-431, 1996

H

[138] Hall AH (1995a): Paraquat usage: environmental fate and effects. In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 17-36, New York: Marcel Dekker

[139] Hall AH (1995b): Paraquat and diquat exposures reported to U.S. poison centers, 1983-1992, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 53-63, New York

[140] Hall AH, and Becker CE, Occupational health and safety considerations in paraquat handling, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 249-266, New York 1995

[141] Hance RJ, Byast TH & Smith PD, (1980): Apparent decomposition of paraquat in soil, Soil Biology and Biochemistry 12(4): 447-448.

[142] Hargreave TB, Gresham GA & Karayannopoulos S (1969): Paraquat poisoning. Postgraduate Medical Journal 45:633-635

[143] Hausburg MA, Dekrey GK, Salmen JJ, Palic MR, and Gardiner CS, Effects of paraquat on development of preimplantation embryos in vivo and in vitro, Reproductive Toxicology 20(2), 239-244, 2005

[144] Hayes WJ, and Laws ER, Quaternary nitrogen compounds, In: Hayes WJ, and Laws ER (eds), Handbook of pesticide toxicology, vol. 3: Classes of pesticides, San Diego, CA: Academic 1991 (quoted by Hall (1995a))

[145] Helling CS, Kaufman DD, and Dieter CT, Weed Science 19, pp. 685ff, 1971 (quoted by Summers (1980))

[146] Hettiarachchi J & Kodithuwakku GC (1989): Self-poisoning in Sri Lanka: factors determining the choice of the poi-soning agents, Human Toxicology 8(6), 507-510.

[147] Heyll K, Vergiftung durch das Herbizid Paraquat: Untersuchungen zum klinischen Verlauf und zur Therapie (PhD thesis), Düsseldorf: Medizinische Fakultät der Universität 1988

[148] Horiuchi N, Oguchi S, Nagami H & Nishigaki Y (2008): Pesticide-related Dermatitis in Saku District, Japan, 1975–2000. International Journal of Occupational and Environmental Health 14:25–34

[149] Hoffer E & Taitelman U, (1989): Exposure to paraquat through skin absorption: clinical and laboratory observations of accidental splashing on healthy skin of agricultural workers, Human Toxicology 8(6): 483-485.

[150] Hoffman DJ, Embryotoxicity and teratogenicity of environmental contaminants to bird eggs, Reviews of Environmental and Contamination Toxicology 115, 40-89, 1990

[151] Holland PT, Glossary of terms relating to pesticides (search: 'margin of safety'), IUPAC Recommendations, 1996

[152] Hoppin JA, Umbach DM, London SJ, Alavanja MC, and Sandler DP, Chemical predictors of wheeze among farmer pesticide applicators in the Agricultural Health Study, American Journal of Respiratory and Critical Care Medicine 165(5), 683-689, 2002

[153] Houze P, Baud FJ, and Scherrmann JM, Toxicokinetics of paraquat, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 161-193, New York 1995

[154] Howard JK, Paraquat spraying: comparative risks from high and low volume application methods, Proceedings of the 10th Asian Conference on Occupational Health, pp. 1-7, Singapore 1982 (quoted by Garnier (1995) and Hall & Becker (1995))

[155] Howard JK, Sabapathy NN, and Whitehead PA, A study on the health of Malaysian plantation workers with particular reference to paraquat spraymen, British Journal of Industrial Medicine 38(2), 110-116, 1981

[156] Hseu ZY, Jien SH, and Cheng SF, Sorption of paraquat on clay components in Taiwan's oxisol, Journal of Environmental Science and Health part B 38(4), 441-449, 2003

[157] Huang J, Quiao F, Zhang L, and Rozelle S, Farm pesticide, rice production and human health, Economy and Environment Program for Southeast Asia (EEPSEA), Ottawa, Canada: IDRC 2000

[158] Hurst P, Safe use in Guatemala - are industry projects effective? Pesticides News 43, 8-9, 1999

[159] Hutchinson G, Daisley H, Simeon D, Simmonds V,Shetty M & Lynn D, (1999): High rates of paraquat-induced suicide in southern Trinidad, Suicide and Life-Threatening Behavior 29(2) 186-191.

I

[160] International Development Research Center (IDRC), Paraquat intoxication (Colombia), Ottawa, Canada 2003

[161] IFCS (2003): Fourth Session of the Intergovernmental Forum on Chemical Safety (IFCS) (Forum IV). Bangkok, Thailand, 1-7 November 2003. Final Report. Geneva.

[162] IPCS (1984): Paraquat and Diquat. Environmental Health Criteria 39. International Programme on Chemical Safety (IPCS), Geneva 1984

[163] IPCS (1991): Paraquat, Health and Safety Guide no. 51. International Programme on Chemical Safety (IPCS). Geneva

[164] IPCS (2001a): Paraquat dichloride. International Programme on Chemical Safety (IPCS). Geneva

[165] IPCS (2004): Guidelines on the prevention of toxic exposures: education and public awareness activities. International Programme on Chemical Safety (IPCS). Geneva

[166] IPM DANIDA (2003): Did you take your poison today? Danish International Development Assistance (DANIDA). Bangkok

[167] IPM DANIDA (2004): "Watch list" or "waiting list?" IPM Newsletter 8, p.3. Danish International Development Assistance (DANIDA)

J

[168] Jansen A-E, Plant protection in coffee: Recommendations for the Common Code for the Coffee Community-Initiative, Deutsche Gesellschaft für Technische Zusammenarbeit (GTZ), Bonn 2005

[169] Jaros F, Acute percutaneous paraquat poisoning, Lancet 1(8058), p. 275, 1978 (quoted by Garnier (1995) and Hall & Becker (1995))

[170] JMPR (1972): Paraquat. (WHO Pesticide Residues Series 2). Joint Meeting on Pesticide Residues (JMPR).

[171] JMPR (1982): Paraquat. PESTICIDE RESIDUES IN FOOD – 1981. FAO PLANT PRODUCTION AND PROTECTION PAPER 42. Joint Meeting on Pesticide Residues (JMPR).

K

[172] Kamel F, Tanner CM, Umbach DM, Hoppin JA, Alavanja MCR, Blair A, Comyns K, Goldman SM, Korell M, Langston JW, Ross GW & Sandler DP (2007): Pesticide Exposure and Self-reported Parkinson's Disease in the Agricultural Health Study. American Journal of Epidemiology 165:364–374

[173] Kamrin MA, Pesticide profiles: Toxicity, environmental impact, and fate, Boca Raton, FL 1997

[174] Kawai M, and Yoshida M, [Exposure of spray operators to paraquat] [Article in Japanese], Nippon Doshu Eisei Zasshi, 28, 353-359, 1981 (quoted by IPCS (1984)).

[175] Keifer M, McConnell R, Pacheco AF, Daniel W & Rosenstock L (1996): Estimating underreported pesticide poisonings in Nicaragua. American Journal of Industrial Medicine 30(2):195-201.

[176] KEMI (2006): Paraquat. Annex: Notification of final regulatory action on paraquat, Sweden. Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade, Chemical Review Committee, Fifth meeting, Rome, 23-27 March, 2009. UNEP/FAO/RC/CRC.5/8. Information submitted in 2006.s

[177] Khan DA, Shabbir S, Majid M, Ahad K, Naqvi TA & Khan FA. (2010): Risk assessment of pesticide exposure on health of Pakistani tobacco farmers. Journal of Exposure Science and Environmental Epidemiology 20(2):196-204

[178] Kishi M, Hirschhorn N, Qjajadisastra M, Satterlee LN, Strowman S, and Dilts, Relationship of pesticide spraying to signs and symptoms in Indonesian farmers, Scandinavian Journal of Work, Environment and Health 21, 124-133, 1995

[179] Kishimoto T, Fujioka H, Yamadori I, Ohke M, Ozaki S, and Kawabata Y, [Lethal paraquat poisoning caused by spraying in a vinyl greenhouse of causing pulmonary fibrosis with a hepatorenal dysfunction][Article in Japanese], Nihon Kokyuki Gakkai Zasshi 36(4), 347-352, 1998

[180] Kishor Atreya (2007): Pesticide use knowledge and practices: A gender differences in Nepal. Environmental Research 104 (2): 305-311.

[181] Kjolholt J, Distribution of pesticides and potential exposure of non-target organisms following application, In: Somerville L, and Walker CH (eds), Pesticide effects on terrestrial wildlife, 33-63, London 1990

[182] Knight BA, and Tomlinson TE, The interaction of paraquat (1:1'-dimethyl 4:4'-dipyridylium dichloride) with mineral soils, Journal of Soil Science 18, 223-243, 1967 (quoted by Summers (1980))

[183] Konradsen F, van der Hoek W, Cole DC, Hutchinson G, Daisley H, Singh S & Eddleston M (2003): Reducing acute poisoning in developing countries - options for restricting the availability of pesticides, Toxicology 192(2-3):249- 261.

[184] Konradsen F, van der Hoek W, Gunnell D & Eddleston M (2005): Missing deaths from pesticide self-poisoning at the IFCS Forum IV, Bulletin of the World Health Organisation 83(2), 157-158.

[185] Konradsen F, Dawson AH, Eddleston M & Gunnell D. (2007): Pesticide self-poisoning: thinking outside the box. Lancet. 369(9557):169-70.

[186] Kookana RS, and Aylmore LA, Retention and release of diquat and paraquat herbicides in soils, Australian Journal of Soil Research 31(1), 97-109, 1993

[187] Kotwica M, Czerczak S, and Rogaczewska A, The pattern of acute poisonings with pesticides in Poland during the periods 1989-1990 and 1994-1995, Przeglad Lekarski 54(10), 689-692, 1997

[188] Kong Y & Zhang J (2010): Access to farming pesticides and risk for suicide in Chinese rural young people. Psychiatry Research 179:217–221

[189] Kurniawan AN (1996): Product stweardship of paraquat in Indonesia, International Archives of Occupational and Environmental Health 68, 516-518.

L

[190] Laborde A (2004): New roles for poison control centres in the developing countries. Toxicology 198(1-3):273-7.

[191] Lajmanovich RC, Izaguirre MF, and Casco VH, Paraquat tolerance and alteration of internal gill structure of Scinax nascia tadpoles (Anura: Hylidae), Archives of Environmental Contamination and Toxicology 34, 364-369, 1998

[192] Láng G, Kufcsák O, Szegletes T, and Nemcsök J, Quantitative distributions of different cholinesterases and inhibition of acetylcholinesterase by metidathion and paraquat in alimentary canal of common carp, General Pharmacology 29(1), 55-59, 1997

[193] Lebailly P, Bouchart V, Baldi I, Lecluse Y, Heutte N, Gislard A & Malas JP (2009): Exposure to Pesticides in Open-field Farming in France. The Annals of Occupational Hygiene (53) 1: 69–81.

[194] Lee K; Park EK; Stoecklin-Marois, Koivunen ME; Gee SJ; Hammock BD; Beckett LA & Schenker MB (2009): Occupational paraquat exposure of agricultural workers in large Costa Rican farms. International Archives of Occupational and Environmental Health 82:4.

[195] Lenaars AA, Moksony F, Lester D & Wenckstern S (2003): The impact of gun control (Bill C-51) on suicide in Canada. Death Studies 27: 103-124.

[196] Lev N & Melamed E (2001): Heredity in Parkinson's disease: new findings. The Israel Medical Association Journal 3 (6):435-8.

[197] Leveridge YR (1998): Pesticide poisoning in Costa Rica during 1996, Veterinary and Human Toxicology 40(1), 42-44.

[198] Leverton K, Cox V, Battershill J & Coggon D (2007): Hospital admission for accidental pesticide poisoning among adults of working age in England, 1998–2003. Clinical Toxicology (45) 5: 594-597

[199] Levin PJ, Klaff LJ, Rose AG, and Ferguson AD, Pulmonary effects of contact exposure to paraquat: A clinical and experimental study, Thorax 34, 150-160, 1979 (quoted by Castro-Gutiérrez (1997), Garnier (1995) and Hall & Becker (1995))

[200] Lewis CP, and Nemery B, Pathophysiology and biochemical mechanisms of the pulmonary toxicty of paraquat, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 107-140, New York: Marcel Dekker 1995

[201] Li S, Crooks PA, Wei X, and de Leon J, Toxicity of dipyridyl compounds and related compounds, Critical Reviews in Toxicology 34(5), 447-460, 2004

[202] Lilley R, Owens D, Horrocks J, House A, Noble R, Bergen H, Hawton K, Casey D, Simkin S, Murphy E, Cooper J & Kapur N (2008): Hospital care and repetition following self-harm: multicentre comparison of self-poisoning and self-injury. British Journal of Psychiatry 192(6):440-5.

[203] Lings S, Pesticide lung: A pilot investigation of fruit growers and farmers during the spraying season, British Journal of Industrial Medicine 39(4), 370-376, 1982

[204] Liou HH, Tsai MC, Chen CJ, Jeng JS, Chang YC, Chen SY, and Chen RC, Environmental risk factors and Parkinson's disease: a case-control study in Taiwan, Neurology 48(6), 1583-1588, 1997

[205] London L & Bailie R (2001): Challenges for improving surveillance for pesticide poisoning: policy implications for developing countries. International Journal of Epidemiology 30, 564-570.

[206] Lubin G, Werbeloff N, Halperin D, Shmushkevitch M, Weiser M & Knobler HY (2010): Decrease in Suicide Rates After a Change of Policy Reducing Access to Firearms in Adolescents: A Naturalistic Epidemiological Study. Suicide and Life-Threatening Behavior 40(5): 421-424.

M

[207] Machado-Neto JG, Matuo T & Matuo YK (1998): Efficiency of safety measures applied to a manual knapsack spray-er for paraquat application to maize (Zea mays L.), Archives of Environmental and Contamination Toxicology 35, 698-701.

[208] Machera K, Goumenou M, Kapetanakis E, Kalamarakis A & Glass CR (2003): Determination of potential dermal and inhalation operator exposure to malathion in greenhouses with the whole body dosimetry method. Annals Occupational Hygiene 47(1):61-70.

[209] Machera K, Tsakirakis A, Charistou A, Anastasiadou P & Glass CR (2009): Dermal exposure of pesticide applicators as a measure of coverall performance under field conditions. Annals Occupational Hygiene 53(6):573-84.

[210] Madeley J (2002a): Unsuitable for use - Profile of paraquat, Pesticides News 56, 3-5

[211] Madeley J (2002b): Paraquat - Syngenta's controversial herbicide, A report written for Berne Declaration, Swedish Society for Nature Conservation, Pesticide Action Network UK, Pesticide Action Network Asia Pacific, Foro Emacis.

[212] Malone JDG, Carmody M, Kheogh B, and O'Dwyer WF, Paraquat poisoning: A review of 19 cases, Journal of the Irish Medical Association 64(405), 59-68, 1971 (quoted by Garnier (1995))

[213] Mann JJ, Apter A, Bertolote J, Beautrais A, Currier D, Haas A, Hegerl U, Lonnqvist J, Malone K, Marusic A, Mehlum L, Patton G, Phillips M, Rutz W, Rihmer Z, Schmidtke A, Shaffer D, Silverman M, Takahashi Y, Varnik A, Wasserman D, Yip P & Hendin H.(2005): Suicide prevention strategies: a systematic review. JAMA 294(16):2064-74.

[214] Marquis JK, Contemporary issues in pesticide toxicology and pharmacology, Basel: Karger 1986

[215] Matthews G, Pesticide use in China - a health and safety concern, Pesticide News 34, p.18, 1996

[216] Matthews G, Wiles T, and Balegue (2003): A survey of pesticide application in Cameroon, Crop Protection 22, 707- 714, 2003

[217] Matthews GA (2008): Attitudes and behaviours regarding use of crop protection products—A survey of more than 8500 smallholders in 26 countries. Crop Protection (27):834–846.

[218] Menet S, Alternatives to paraquat (unpublished project paper), Zurich 2002

[219] Mercado C, Personal communication by Camelia Mercado (Cooperativa de Parceleros y pequenos Productores de Banano - ASOPROBAN) to Berne Declaration, August 2002

[220] Meredith T, and Vale JA, Treatment of paraquat poisoning: gastrointestinal decontamination, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 297-313, New York: Marcel Dekker 1995

[221] Mordaunt CJ, Gevao B, Jones KC, and Semple KT, Formation of non-extractable pesticide residues: observations on compound differences, measurement and regulatory issues, Environmental Pollution 133(1), 25-34, 2005

[222] Morshed MM, Omar D, Mohamad R, Wahed S & Rahman MA (2010): Airborne paraquat measurement and its exposure in treated field environment. International Journal of Agriculture & Biology 12: 679–684.

[223] Mowbray DL, Pesticide poisoning in Papua New Guinea and the South Pacific, Papua New Guinea Medical Journal 29(2), 131-141, 1986

[224] Muioz Piia C, and Forcada SA, Effects of an environmental tax on pesticides in Mexico, UNEP Industry and Environment 27(2-3), 33-36, Paris 2004

[225] Murphy H, Sanusi A, Dilts A, Djajadisastra M, Hirschhorn N, and Yuliantiningsih S, Health effects of pesticides use among Indonesian women farmers: part I: Exposure and acute health effects, Journal of Agromedicine 6(3), 61-85, 1999

[226] Murray DL & Taylor PL (2000): Claim no easy victories: Assessing the pesticide industry's global Safe Use campaign, World Development 28(1), 1735-1749.

[227] Murray DL, Wesseling C, Keifer M, Corriols M &Henao S (2002): Surveillance of pesticide-related illness in the developing world: putting the data to work. International Journal of Occupational and Environmental Health 8(3):243-8.

N

[228] Nagami H, Nishigaki Y, Matsushima S, Matsushita T, Asanuma S, Yajima N, Usuda M & Hirosawa M (2005): Hospital-based survey of pesticide poisoning in Japan, 1998-2002. International Journal of Occupational and Environmental Health 11(2):180-184.

[229] Nagami H, Nishigaki Y, Matsushima S & Yajima N (2007): Paraquat poisoning in Japan: A Hospital based Survey. Japanese Journal for Rural Medicine 2:85-92.

[230] Nemcsok et al, Accumulation of pesticides on the organs of carp (Cyprinus carpio L.) at 4°C and 20°C, Bulletin of Environmental Contamination and Toxicology 39(3), 370-378, 1987 (quoted by Hall (1995))

[231] Neubert S, and Knirsch J, Herbicide resistant crops - Appropriate Technology for the Third World? In: Dinham B, Growing food security: challenging the link between pesticides and access to food, 22-24, London: The Pesticides Trust/PAN, 1996 (and reference therein: Gworgwor 1989)

[232] Newhouse M, McEvoy D, and Rosenthal D, Percutaneous paraquat absorption, Archives of Dermatology 114(10), 1516-1519, 1978 (quoted by Garnier (1995) and Hall & Becker (1995))

[233] Ngowi AVF, Mbise TJ, Ijani ASM, London L & Ajayic OC (2007): Smallholder vegetable farmers in Northern Tanzania: Pesticides use practices, perceptions, cost and health effects. Crop Protection 26:1617–1624.

[234] NIOSH (1996): NIOSH chemical listing and documentation of revised IDLH values (as of 3/1/95): Paraquat National Institute for Occupational Safety and Health (NIOSH). Atlanta. USA

[235] Nishimoto RK, Weed management in coffee plantations, In: UN Food and Agriculture Organisation, Weed management in developing countries, Rome: FAO 1994

[236] NLM (1994): Paraquat. Hazardous Substances Data Bank. National Library of Medicine (NLM). Bethesda, MD.

[237] NRDC (2004): Hidden danger: environmental health threats in the Latino community, Natural Resources Defense Council (NRDC), New York, NY 2004

[238] Ntow WJ, Gijzen HJ, Kelderman P & Drechsel P (2006): Farmer perceptions and pesticide use practices in vegetable production in Ghana. Pest Management Science 62 (4): 356–365.

O

[239] Ochoa Gomez FJ, and Gil Paraiso A, [Fatal poisoning with paraquat: report of a new case] [Article in Spanish], Anales de Medicina Interna 10(7), 349-350, 1993

[240] OECD (1995): Development Assistance Committee, Guidelines for Aid Agencies on Pest and Pesticide Management, DAC Guidelines on Aid and Environment no. 6. Organisation for Economic Cooperation and Development (OECD)

[241] OECD (2005): The assessment of persistency and bio-accumulation in the Pesticide Registration Frameworks within the OECD region. Organisation for Economic Co-operation and Development (OECD).

[242] Ohayo-Mitoko GJ, Kromhout H, Karumba PN, and Boleij JS, Identification of pesticide exposure among Kenyan agricultural workers using empirical modelling, Annals of Occupational Hygiene 43(8), 519-525, 1999

[243] Ong ML & Glew S (1989): Clinical Toxicology. Paraquat poisoning: per vagina. Postgraduate Medical Journal 65: 835–836.

[244] OPS/OMS (2001b): Exposiciön dérmica a plaguicidas en un bananera, San José, Costa Rica: OPS/Organizaciön Mundial de la Salud (OMS): Proyecto PLAGSALUD, Organizaciön Panamericana de la Salud (OPS).

[245] OPS/OMS (2002a): Intoxicaciones por plaguicidas en Costa Rica: Informe epide-miolögico 2001, San José, Costa Rica: OPS/Organizaciön Mundial de la Salud (OMS): Proyecto PLAGSALUD, Organizaciön Panamericana de la Salud (OPS),

[246] OPS/OMS (2002b): Proyecto PLAGSALUD Costa Rica: Memoria fase II, San José, Costa Rica: OPS/Organizaciön Mundial de la Salud (OMS): Proyecto PLAGSALUD, Organizaciön Panamericana de la Salud (OPS),

[247] OPS/OMS (2002c): Subregistro de las intoxicaciones por plaguicidas, San José, Costa Rica: OPS/Organizaciön Mundial de la Salud (OMS): Proyecto PLAGSALUD, Organizaciön Panamericana de la Salud (OPS),

[248] Osano O, Oladimeji AA, Kraak MH, and Admiraal W, Teratogenic effects of amitraz, 2,4-dimethylaniline, and paraquat on developing frog (Xenopus) embryos, Archives of Environmental Contamination and Toxicology 43, 42-49, 2002

[249] Owens D, Wood C, Greenwood DC, Hughes T & Dennis M (2005): Mortality and suicide after non-fatal self-poisoning: 16-year outcome study. British Journal of Psychiatry. 87:470-5.

P

[250] Palis FG, Flor RJ, Warburton H & Hossain M 2006): Our farmers at risk: behaviour and belief system in pesticide safety. Journal of Public Health 28(1): 43-48.

[251] PANAP (2010): Paraquat Monograph. PAN Asia and the Pacific (PANAP)

[252] PANNA (1995): Demise of the Dirty Dozen 1995 chart, Pesticide Action Network of North America (PANNA). San Francisco. http://www.panna.org/files/dirtyDozenChart.dv.html

[253] PAN UK (1998): Pest management notes no. 9, Growing coffee with IPM. Pesticide Action Network (PAN) United Kingdom (UK).

[254] Papiris SA, Maniati MA, Kyriakidis V, and Costantopoulos SH, Pulmonary damage due to paraquat poisoning through skin absorption, Respiration 62(2), 101-103, 1995

[255] Parellada M, Saiz P, Moreno D, Vidal J, Llorente C, Alvarez M, García-Portilla P, Ruiz-Sancho A, Arango C & Bobes J (2008): Is attempted suicide different in adolescent and adults?. Psychiatry Research 157:131–137.

[256] Partanen T, Chaves J, Wesseling C, Chaverri F, Monge P, Ruepert C, Aragon A, Kogevinas M, Hogstedt C, and Kauppinen T, Workplace Carcinogen and pesticide exposures in Costa Rica, International Journal of Environmental and Occupational Health 9(2), 104-111, 2003

[257] Pasi A (1978): The toxicology of paraquat, diquat and morfamquat, Berne: Hans Huber Publishers.

[258] Peiró AM, Zapater P, Alenda C, Ramírez A, Gutiérrez A, Pérez-Mateo M & Such J (2007): Hepatotoxicity Related to Paraquat and Diquat Absorption Through Intact Skin. Digestive Diseases and Sciences 52 (11)3282-4.

[259] Penagos HG, Contact dermatitis caused by pesticides among banana plantation workers in Panama, Interna-tional Journal of Occupational and Environmental Health 8 (1), 14-18, 2002

[260] Petrovitch H, Ross GW, Abbott RD, Sanderson WT, Sharp DS, Tanner CM, Masaki KH, Blanchette PL, Popper JS, Foley D, Launer L & White LR (2002): Plantation Work and Risk of Parkinson Disease in a Population-Based Longitudinal Study. Archives of Neurology 59:1787-1792.

[261] PIC Secretariat (2008): Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade. Text and Annexes (Revised in 2008). Rotterdam Convention Secretariat (PIC Secretariat).

[262] PIC Secretariat (2010): Pilot Study on Agricultural Pesticide Poisoning in Burkina Faso. Final Report.Secretariat of the Rotterdam Convention. On the Prior Informed Consent (PIC) Procedure for Certain Hazardous Chemicals and Pesticides in International Trade.

[263] Pingali PL, and Gerpacio RV, Toward reduced pesticide use for cereal crops in Asia, In: Lutz E (ed), Agriculture and the environment: perspectives on sustainable development, Washington, D.C.: The World Bank 1998

[264] Plestina R, Prevention, diagnosis and treatment of insecticide poisoning (unpublished World Health Organisation document WHO/VBC/84.889), Geneva: WHO 1984 (Obtainable from the International Programme on Chemical Safety), Geneva, Switzerland

[265] Pond SM, Treatment of paraquat poisoning, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 325-334, New York: Marcel Dekker 1995

[266] Porteous F, Killham K, and Meharg A, Use of a lux-marked rhizobacterium as a biosensor to assess changes in rhizosphere C flow due to pollutant stress, Chemosphere 41(10), 1549-1554, 2000

[267] Prakasam A, Sethupathy S, and Lalitha S, Plasma and RBCs antioxidant status in occupational male pesticide sprayers, Clinica Chimica Acta 310(2), 107-112, 2001

[268] Pronczuk de Garbino J (1995): Epidemiology of paraquat poisoning, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 37-51, New York: Marcel Dekker

[269] Protano C, Guidotti M & Vitali M (2009): Performance of different work clothing types for reducing skin exposure to pesticides during open field treatment. Bulletin of Environmental Contamination and Toxicology 83(1):115-9.

[270] Proudfoot AT & Dougall H (1988): Poisoning treatment centre admissions following acute incidents involving pesticides. Human Toxicology 7(3):255-258.

[271] Przybylska A, [Intoxications caused by chemicals for plant protection in Poland in 2000] [Article in Polish], Przeglad Epidemiologiczny 56(2), 311-317, 2002

[272] Przybylska A, [Intoxications caused by plant protection chemicals in 1997] [Article in Polish] Przeglad Epide-miologiczny 53(1-2), 121-128, 1999

Q

[273] Quijano R (2002): Peasant movement on the Philippines, Kamukhaan: report on a poisoned village.

R

[274] Racke KD, Skidmore MW, Hamilton DJ, Unsworth JB, Miyamoto J (1997): Cohen SZ, Pesticide fate in tropical soils, Pure and Applied Chemistry 69(6), 1349-1371.

[275] Recena MCP, Pires DX, Caldas ED (2006): Acute poisoning with pesticides in the state of Mato Grosso do Sul, Brazil. Science of the Total Environment 357:88– 95.

[276] Resudarmo B, Integrated Pest Management in Indonesia: the cost of chemicals, Ottawa, Canada: International Development Research Center 2000

[277] Ribas G, Surralles J, Carbonell E, Xamena N, Creus A, and Marcos R, Genotoxic evaluation of the herbicide paraquat in cultured human lymphocytes, Teratogenisis, Carcinogenisis, and Mutagenisis 17(6), 339-347, 1997-98

[278] Ritz BR, Manthripragada AD, Costello S, Lincoln SJ, Farrer MJ, Cockburn M & Bronstein J (2009): Dopamine Transporter Genetic Variants and Pesticides in Parkinson's Disease. Environmental Health Perspectives 117 (6).

[279] Riley D, Tucker BV & Wilkinson W (1976): Unavailability of Bound Paraquat Residue in Soil. In Bound and Conjugated Pesticide Residues. Kaufman DD, Still GG, Paulson GD & Bandal SK (eds.). Symposium Series 29. American Chemical Society. Washington. pp 301-353.

[280] Roberts DM, Karunarathna A, Buckley NA, Manuweera G, Sheriff MH, and Eddleston M, Influence of pesticide regulation on acute poisoning deaths in Sri Lanka, Bulletin of the World Health Organisation 81 (11), 789-798, 2003

[281] Roberts TR, Dyson JS, and Lane MC, Deactivation of the biological activity of paraquat in the soil environment: a review of the long-term environmental fate, Journal of Agriculture and Food Chemistry 50(13), 3623-3631, 2002

[282] Rodricks JV, Calculated risks: understanding the toxicity and human health risks of chemicals in our environment, Cambridge: CUP 1992

[283] Rojo AI, Cavada C, de Sagarra MR &, Cuadrado A (2007): Chronic inhalation of rotenone or paraquat does not induce Parkinson's disease symptoms in mice or rats. Experimental Neurology 208:120–126

[284] Ronnen M, Klin B, Suster S, Mixed diquat/paraquat-induced burns, International Journal of Dermatology 34(1), 23-25, 1995

[285] Rose MS, and Smith LS, The relevance of paraquat accumulation by tissues, In: Autor AP (ed), Biochemical mechanisms of paraquat toxicity, 71-91, New York: Academic Press 1977

[286] Rosencranz A, Roblin S & Balloffet N (2009): Doling out Environmental Justice to Nicaraguan Banana Workers: The Jose Adolfo Tellez v. Dole Food Company Litigation in the U.S. Courts. 3 Golden Gate U. Envtl. L.J. http://digitalcommons.law.ggu.edu/gguelj/vol3/iss1/7

[287] Rother HA (2008): South African farm workers' interpretation of risk assessment data expressed as pictograms on pesticide labels. Environmental Research 108:419–427.

[288] Rutz R, and Krieger RI (1992): Exposure to pesticide mixer/loaders and applicators in California, Reviews of Environ-mental Contamination and Toxicology 129, 121-139.

S

[289] Sáenz ME, Di Marzio WD, Alberdi JL, and Tortorelli MC, Algal growth recovery studies after paraquat exposure, Bulletin of Contamination and Toxicology 66, 263-268 2001

[290] Santus P, Sola A, Carlucci P, Fumagalli F, Di Gennaro A, Mondoni M, Carnini C, Centanni S, and Sala A, lipid peroxidation and 5-lipoxygenase activity in chronic obstructive pulmonary disease, American Journal of Respiratory and Critical Care Medicine 171, 838-843, 2004

[291] Schenker MB, Stoecklin M, Lee K, Lupercio R, Zeballos RJ, Enright P, Hennessy T, and Beckett LA, Pulmonary function and exercise-associated changes with chronic low-level paraquat exposure, American Journal of Respiratory and Critical Care Medicine 170(7), 773-779, 2004

[292] Segen JC (ed), The dictionary of modern medicine, Basel: Roche 1992

[293] Seiber JN, Woodrow JE, Hermann BW, and Sanders P, Determination of airborne residues from four harvest aid chemicals (DEF, folex, cacodylates, and paraquat) at treated San Joaquin Valley cotton field sites, as a measure of potential human exposure, document HS-1326 (California EPA, Department for Pesticide Regulation), Davis, California: University of California 1983

[294] Semple S, Dermal exposure to chemicals in the workplace: just how important is skin absorption? Occupational and Environmental Medicine 61(4), 376-382, 2004

[295] Senanayake N, Gurunathan G, Hart TB, Amerasinghe P, Babapulle M, Ellapola SB, Udupihille M, and Basanayake V, An epidemiological study of the health of Sri Lankan tea plantation workers associated with long term exposure to paraquat, British Journal of Industrial Medicine 50(3), 257-263, 1993

[296] Seok SJ, Gil HW, Jeong DS, Yang JO, Lee EY & Hong SY (2009): Paraquat Intoxication in Subjects Who Attempt Suicide: Why They Chose Paraquat. The Korean Journal of Internal Medicine 24:247-251

[297] Sirajuddin H, Roslinah A, Rampal KG, Kuppusamy I, Rohna R, Aziz M, Aw TC & Beach JR (2002): Notification of occupational and work-related diseases and poisonings in Malaysia, 1997-1998. Medical Journal of Malaysia 56(1): 25-31.

[298] Smith EA, and Mayfield CI, Paraquat: determination, degradation and mobility in soil, Water, Air and Soil Pollution 9, 439-452, 1978

[299] Smith EA, and Oehme FW, A review of selected herbicides and their toxicities, Veterinary and Human Toxicology 33(6), 596-608, 1991

[300] Smith JG, Paraquat poisoning by skin absorption: a review, Human Toxicology 7(1), 15-19, 1988

[301] Snelder DJ , Masipiqueña MD & de Snoo GR (2008): Risk assessment of pesticide usage by smallholder farmers in the Cagayan Valley (Philippines). Crop Protection, 27 (3-5):747-762.

[302] Spark KM, and Swift RS, Effect of soil composition and dissolved organic matter on pesticide sorption, Science of the Total Environment 298(1-3), 147-161, 2002

[303] Spruit O, and van Puijvelde, Evaluation of the protective equipment used during herbicide application on banana plantations, Internal report 1998-304, Wageningen Agricultural University 1998 (quoted by Wesseling et al (2005))

[304] Staiff DC, Comer SW, Armstrong JF, and Wolfe HR, Exposure to the herbicide, paraquat, Bulletin of Environmental Contamination and Toxicology 14, 334-340, 1975 (quoted by Garnier (1995))

[305] Stephens BG & Moormeister SK (1998): Homicidal poisoning by paraquat. Comment in: American Journal of Forensic Medicine & Pathology 19(3):294-5.

[306] Stevens JT, and Sumner DD, Herbicides, In: Hayes WJ, and Laws ER (eds), Handbook of pesticide toxicology, 1356-1376, San Diego, CA 1991 (quoted by Wesseling et al (1993))

[307] Stratta P, Mazzucco P, Griva S, Tetta C, and Monga G, Immune-mediated glomerulonephritis after exposure to paraquat, Nephron 48(2), 138-141, 1988 (quoted by Hall & Becker (1995) and Garnier (1995))

[308] Srinivas Rao Ch, Venkateswarlu V, Surender T, Eddleston M & Buckley NA (2005): Pesticide poisoning in south India: opportunities for prevention and improved medical management. Tropical Medicine and International Health 10(6):581-8.

[309] Summers LA (1980): The bipyridinium herbicides, London: Academic Press.

[310] Swan AAB, Exposure of spray operators to paraquat, British Journal of Industrial Medicine 26, 322-329,1969

[311] Syngena (2005): Gramoxone (product label). Syngenta Crop Protection Co., Ltd., Thailand 2005

[312] Syngenta (2010): Gramoxone Inteon. Specimen Label EPA SCP 1217A-L1D 0610). Syngenta Crop Protection.

[313] Syngenta Agro, S.A. de C.V., Gramoxone (product label), Del Valle, México Distrito Federal, In: Thomson PL, Diccionario de especialidades agroquimicas, México 2004

[314] Syngenta International AG, Product stewardship: Gramoxone, 2005

[315] Syngenta International AG, The responsible stewardship of our crop protection products, Basel 2003

T

[316] Tanner CM, Kamel F, Ross GW, Hoppin JA, Goldman SM, Korell M, Marras C, Bhudhikanok GS, Kasten M, Chade AR, Comyns K, Richards MB, Meng C, Priestley B, Fernandez HH, Cambi F, Umbach DM, Blair A, Sandler DP, Langston JW (2011): Rotenone, Paraquat and Parkinson's Disease. Environmental Health Perspective 2011 Jan 26

[317] Tanner CM, Ross GW, Jewell SA, Hauser RA, Jankovic J, Factor SA.Bressman S, Deligtisch A, Marras C, Lyons KE, Bhudhikanok GS, Roucoux DF, Meng C, Abbott RD & Langston JW (2009): Occupation and Risk of Parkinsonism. A Multicenter Case-Control Study. Archives of Neurology 66 (9):1106-1113

[318] Taylor PJ, Salm P & Pillans PI (2001): A detection scheme for paraquat poisoning: validation and a five-year experience in Australia. Journal of Analytical Toxicology 25(6):456-60.

[319] Tenaganita, and Pesticide Action Network Asia and the Pacific (PANAP), Poisoned and silenced: a study of pesticide poisoning in the plantations, Kuala Lumpur, Malaysia 2002

[320] Tenaganita, and Pesticide Action Network Asia and the Pacific (PANAP), Victims without voice: a study of women pesticide workers in Malaysia, Kuala Lumpur, Malaysia 1992

[321] Terry PJ, Weed management in bananas and plantains, In: UN Food and Agriculture Organisation, Weed management in developing countries, Rome: FAO 1994

[322] Texeira H, Proença P, Alvarenga M, Oliveira M, Marques E, and Vieira D, Pesticide intoxications in the Centre of Portugal, Forensic Science International 143(2-3), 199-204, 2004

[323] Thompson JP, Casey PB & Vale JA (1995a) Pesticide incidents reported to the Health and Safety Executive 1989/90- 1991/92, Human and Experimental Toxicology 14(8), 630-633.

[324] Thompson JP, Casey PB & Vale JA (1995b): Deaths from pesticide poisoning in England and Wales 1990-1991, Human and Experimental Toxicology 14(5), 437-445.

[325] Tomlin CDS (ed.) (2003): Pesticide Manual: A World Compendium of Pesticides. British Crop Protection Council.

[326] Tortorelli MC, Hernández DA, Rey Vazquez G, and Salibián A, Effects of paraquat on mortality and cardiorespiratory function of catfish Plecostomus commersoni, Archives of Environmental Contamination and Toxicology 19, 253.259, 1989

[327] Tsukamoto M, Tampo Y, Sawada M, and Yonaha M, Paraquat-induced membrane dysfunction in pulmonary microvascular endothelial cells, Toxicology and Applied Pharmacology 86(3), 102-109, 2000

[328] Tungsanga K, Chusilp S, Israsena S & Sitprija V(1983): Paraquat poisoning: evidence of systemic toxicity after dermal exposure. Postgraduate Medical Journal 59:338–539

U

[329] UN DESA (2002): Key commitments, targets and time-tables from the Johannesburg Plan of Implementation United Nations Department of Economic and Social Affairs (UNDESA). New York.

[330] UN DESA (2004): Plan of Implementation of the World Summit on Sustainable Development, United Nations Department of Economic and Social Affairs. New York .

[331] UNEP (2004a): Childhood pesticide poisoning: information for advocacy and action. Geneva, Chemicals Programme of the United Nations Environment Programme (UNEP), Food and Agriculture Organisation, World Health Organisation, 2004 (http://www.who.int/entity/ceh/publications/pestpoisoning.pdf).

[332] UNEP (2004b): Making agriculture more sustainable: trends and challenges, UNEP Industry and Environment 27(4) United Nations Environment Programme (UNEP).

[333] UNEP (2006): Strategic Approach to International Chemicals Management. SAICM texts and resolutions of the International Conference on Chemicals Management. United Nations Environment Programme (UNEP).

[334] UNEP (2010): Consolidated List of Products whose Consumption and/ or Sale have been Banned, Withdrawn, Severely Restricted or Not Approved by Governments Contribution by UNEP covering Pesticides and Industrial Chemicals. United Nations Environment Programme (UNEP).

[335] UNESCO (1999): Summary Record of the 21st Meeting held at the Palais des Nations, Geneva, on Thursday, 8 April 1999, Commission on Human Rights, 55th session, document E/CN.4/1999/SR.21, item 65. United Nations Economic and Social Council (UNESCO). New York .

[336] US Department of Agriculture (USDA) Agricultural Research Service, ARS pesticide properties list, 1995

[337] US EPA (1988): Paraquat, In: US EPA, Pesticide fact handbook, 596-605, Park Ridge, NJ: Noyes Data Corp. 1988 (quoted by Hall (1995)) US Environmental Protection Agency (EPA).

[338] US EPA (1991): Integrated Risk Information System, Paraquat: Oral RfD assessment, Washington, D.C. (1987, revised 1991). US Environmental Protection Agency (EPA).

[339] US EPA (1993): Integrated Risk Information System, Paraquat: Carcinogenicity assessment, Washington, D.C. (1987, revised 1993). US Environmental Protection Agency (US EPA). http://www.epa.gov/iris/subst/0183.htm assessed 17.03.2011

[340] US EPA (1997a): Reregistration Eligibility Decision (RED): Paraquat Dichloride. US Environmental Protection Agency (EPA).

[341] US EPA (1997b): R.E.D. Facts: Paraquat Dichloride (fact sheet). US Environmental Protection Agency (EPA).

[342] US EPA (2010): Chemicals Evaluated for Carcinogenic Potential. August 2010. US Environmental Protection Agency (US EPA). Office of Pesticide Programs.

[343] US NTP (1995): Hazard information on toxic chemicals added to EPCRA section 313 under chemical expansion, table 3: chronic (non-cancer) toxicity, US National Toxicology Program (NTP).

[344] US NTP (2005): Testing status: methyl viologen. US National Toxicology Program (NTP). North Carolina 2005

V

[345] Vale JA, Meredith TJ, and Buckley BM, Paraquat poisoning: clinical features and immediate general management, Human Toxicology 6(1), 41-47, 1987

[346] van der Hoek W, and Konradsen F, Risk factors for acute pesticide poisoning in Sri Lanka, Tropical Medicine and International Health 10(6), 589-596, 2005

[347] Van Hemmen JJ, Groeneveld CN, Van Drooge H, Van Haelst AG, Schipper AH, and van der Jagt KE, Risk assessment of worker and residential exposure to pesticides: conclusions and recommendations, Annals of Occupational Hygiene 45(1001), S171-S174, 2001

[348] van Wendel de Joode BN, de Graaf IA, Wesseling C & Kromhout H (1996): Paraquat exposure of knapsack applicators on banana plantations in Costa Rica. International Journal of Occupational and Environmental Health 2, 294-304.

[349] Vidal JL, Vega AB, Lopez FJ, and Frenich AG (2004): Application of internal quality control to the analysis of quaternary ammonium compounds in surface and groundwater from Andalusia (Spain) by liquid chromatography with mass spectrometry, Journal of Chromatography part A 1050(2), 179-184.

[350] Vijayakumar (2007): Suicide and its prevention: The urgent need in India. Indian Journal of Psychiatry 49:81–4.

[351] Villaplana J, Azon A, Romaguera C, Lecha M, Phototoxic contact dermatitis with toxic hepatitis due to the per-cutaneous absorption of paraquat, Contact Dermatitis 29(3), 163-164, 1993

[352] Vismara C, Battista VV, Vailati G & Bacchetta R (2000): Paraquat induced embryotoxicity on Xenopus laevis development. Aquatic Toxicology 49:171–179.

[353] Vitali M, Protano C, Del Monte A, Ensabella F & Guidotti M (2009): Operative Modalities and Exposure to Pesticides During Open Field Treatments Among a Group of Agricultural Subcontractors. Archives of Environmental Contamination and Toxicology 57(1):193-202.

[354] Vlahodimos KP, The global crop protection industry strives to bring 'safe use' to the developing world, Pesticide Outlook 5, 194-197, 1999

[355] Volcafe, Personal communication to F. Meienberg, April 2003

W

[356] Wagenet LP, Lemley AT, and Wagenet RT, A review of physico-chemical parameters related to the soil groundwater fate of selected pesticides used in New York State: paraquat, NY: Cornell University 1985

[357] Waichman AV, Römbke J, Ribeiro MOA & Nailson CSN (2002): Review Articles Use and fate of pesticides in the Amazon State, Brazil Risk to human health and the environment. Environmental Science and Pollution Research 9 (6): 423-428, DOI: 10.1007/BF02987596

[358] Waight JJ, Fatal percutaneous paraquat poisoning [correspondence], Journal of the American Medical Association 242, p. 472, 1979 (quoted by Garnier 1995 and Hall & Becker (1995))

[359] Wang Z, Wang Z & Xing J (2011): The quantitative analysis of paraquat in biological samples by liquid chromatography electrospray ionization-mass spectrometry. Journal of Analytical Toxicology 35(1):23-7.

[360] Watterson A, Pesticide users' health and safety handbook, an international guide, Aldershot, England 1988

[361] Weber JB, Best JA, and Gonese JU, Bioavailability and bioactivity of sorbed organic chemicals: quaternary ammonium compounds, in: Linn DT et al (ed), Sorption and degradation of pesticides and organic chemicals in soil, 158-164, SSSA Special Publication no. 32, Madison, Wi: Soil Science Society of America 1993

[362] Weber JB & Scott DS (1966): Availability of a Cationic Herbicide Adsorbed on Clay Minerals to Cucumber Seedlings. Science (3):1400-1402. [DOI:10.1126/science.152.3727.1400]

[363] Wegmann E, Untersuchungen über die Paraquatakkumulation in limnischen Sedimenten am Beispiel des Schwentineeinzugsgebietes: Ein Beitrag zum Problem der chemischen Grabenentkrautung (PhD thesis), Kiel: Christian-Albrechts-Universität 1977

[364] Weinbaum Z, Samuels SJ, and Schenker MB, Risk factors for occupational illnesses associated with the use of paraquat (1,1'-dimethyl-4,4'-bipyridylium dichloride) in California, Archives of Environmental Health 50(5), 341-348, 1995

[365] Wesseling C, Ahlbom A, Antich D, Rodriguez AC, and Castro R, Cancer in banana plantation workers in Costa Rica, International Journal of Epidemiology 25(6), 1125-1131, 1996

[366] Wesseling C, Castillo L, and Elinder C-G (1993): Pesticide poisonings in Costa Rica, Scandinavian Journal of Work, Environment and Health 19, 227-235.

[367] Wesseling C, Corriols M, and Bravo V (2005): Acute pesticide poisoning and pesticide registration in Central America. Toxicology and Applied Pharmacology 207 (2 Suppl 1), 697-705.

[368] Wesseling C, Hogstedt C, Picado A, & Johansson L (1997): Unintentional fatal paraquat poisonings among agri-cultural workers in Costa Rica: a report of fifteen cases, American Journal of Industrial Medicine 32 (5): 433-441

[369] Wesseling C, van Wendel de Joode B & Monge P (2001b): Pesticide-related illness among banana workers in Costa Rica: A comparison between 1993 and 1996, International Journal of Occupational and Environmental Health (7) 90-97.

[370] Wesseling C, van Wendel de Joode B, Ruepert C, Leon C, Monge P, Hermosillo H, and Partanen TJ (2001a): Paraquat in developing countries, International Journal of Occupational and Environmental Health 7(4): 275-286.

[371] Wester RC, Maibach HI, Bucks DA, and Aufrere MB, In vivo percutaneous absorption of paraquat from hand, leg, and forearm of humans, Journal of Toxicology and Environmental Health 14(5-6), 759-762, 1984

[372] Westerlund M, Hoffer B & Olson L (2010): Parkinson's disease: Exit toxins, enter genetics. Progress in Neurobiology 90(2):146-56.

[373] Whitaker MJ, The handling and use of paraquat by Malaysian rubber and oil palm smallholders, Journal of Plant Protection in the Tropics 6, 231-249, 1989 (quoted by Matthews et al (2003))

[374] WHO & UNEP (1990): Public health impact of pesticides used in agriculture. World Health Organisation (WHO) & United Nations Environment Program (UNEP), Geneva.

[375] WHO (2002): World Report on Violence and Health. Chapter 7. World Health Organisation (WHO).

[376] WHO (2004): Atlas: country resources for neurological disorders 2004. World Health Organisation (WHO) http://www.who.int/mental_health/neurology/neurogy_atlas_lr.pdf

[377] WHO (2010): The WHO Recommended Classification of Pesticides by Hazard and Guidelines to Classification 2010. World Health Organisation (WHO). Geneva.

[378] Williams RL, Aston RS & Krieger RI (2004): Perspiration increased human pesticide absorption following surface contact during an indoor scripted activity program, Journal of Exposure Analysis and Environmental Epidemiology 14(2), 129-136.

[379] Williamson S, Ball A & Pretty J (2008): Trends in pesticide use and drivers for safer pest management in four African countries. Crop Protection 27:1327– 1334.

[380] Wilks MF, Tomenson JA, Fernando R, Ariyananda PL, Berry DJ, Buckley NA, Gawarammana IB, Jayamanne S, Gunnell D & Dawson A (2011): Formulation changes and time trends in outcome following paraquat ingestion in Sri Lanka. Clinical Toxicology:49: 21–28.

[381] Winchester JF, History of paraquat intoxication, In: Bismuth C, and Hall AH (eds), Paraquat poisoning: mechanisms, prevention, treatment, 1-16, New York: Marcel Dekker 1995

[382] Wohlfahrt DJ, Fatal paraquat poisonings after skin absorption, Medical Journal of Australia 1(12), 512-513, 1982 (quoted by Garnier (1995) and Hall & Becker (1995))

[383] Wohlfahrt DJ, Paraquat poisoning in Papua New Guinea, Papua New Guinea Medical Journal 24(3), 164-168, 1981

[384] Wojeck GA, Price JF, Nigg HN, and Stamper JH, Worker exposure to paraquat and diquat, Archives of Environmental Contamination and Toxicology 12, pp. 65ff, 1983

[385] WSC (2009): International survey of herbicide resistant weeds. (www.weedscience.org) Select 'herbicide mode of action' D (Bipyiridyliums). Weed Science.

Y

[386] Yamashita M, Yamashita M & Ando Y (2000): A long-term follow-up of lung function in survivors of paraquat poisoning, Human and Experimental Toxicology 19(2), 99-103.

[387] Yan AS, Keuk K & San S (2002): A survey of the health effects of Pesticides Survey as part of a farmer field school exercise in Cambodia, In: Murphy HH, Summary of farmer health studies, Conference on Health Effects of Pesticides, Penang, Malaysia, 18-20 March, FAO Programme for Community IPM (CIPM) in Asia.

[388] Yoon KC, Im SK, Kim JC, Yoon KW & Choi SK (2009): Prognosis of paraquat-induced ocular surface injury: therapeutic effect of amniotic membrane transplantation. Cornea 28(5):520-3.

Z

[389] Zahl DL & Hawton K (2004): Repetition of deliberate self-harm and subsequent suicide risk: long-term follow-up study of 11 583 patients.The British Journal of Psychiatry 185: 70 - 75.

[390] Zenz C, Occupational medicine, third edition, St. Louis, MO 1994

[391] Zinn C (1995): South Pacific leads the world in rates of youth suicide (News). BMJ 311, 830.

Annex I - Documentation of unsafe pesticide use practices

Africa

A study on children working as pesticide applicators in Egypt showed that, although some personal protective equipment (PPE) was available at the local agricultural office and the applicators have access to it, there were no regulations in **Egypt** requiring PPE, no formal training on its use was offered [conducted], and PPE was not commonly used by the applicators. Some wore dust masks and safety glasses to prevent splashes, but this was not the norm in 2005. The main routes of exposure were dermal exposure and inhalation. The applicator groups had significantly impaired neurobehavioral performance on all of the measures compared to the control groups (Abdel Rasoul et al. 2008).

A survey of spraying equipment in **Cameroon**, where paraquat and glyphosate were the most commonly used herbicides, found that lever–operated knapsack sprayers predominated in two areas, while in a drier area it was mostly CDA (controlled droplet application) sprayers that were used. Leakages were reported by users of lever–operated knapsack sprayers on several different parts of the sprayer, with faults occurring mainly at the nozzle (blockage) and trigger valve. Leakage increased as the sprayers aged. About 25% of sprayers were considered by users to be in good condition and another 25% to be well–maintained. Less than a quarter of all farmers had spare parts and newer sprayers were generally on larger farms and plantations. The sprayers of most small–scale farmers were in a poor condition and over 85% of these farmers did not use protective clothing (Matthews et al. 2003).

In **Kenya** pesticide poisoning occurred despite use of personal protection. Protective equipment was either not used properly, it seems, or was soaked with pesticides during spraying, resulting in dermal exposure (OhayoMitoko et al 1999). Most clothing was made of cotton that soaked up pesticides. Wearing boots only improved the level of protection when combined with a coverall made of heavier cloth (Ohayo-Mitoko et al. 1999).

Fiftysix per cent of smalls–cale cotton farmers in **Zimbabwe** reported pesticide related health problems. Protective equipment did not present a panacea to health risks from pesticides as it was found that protective practices (e.g. wearing a coverall) explained only a small share of total variance of health effects (Angehrn 1996). The use of protective equipment was low, partly because the benefits of such equipment did not seem overwhelming, and it was connected with discomfort, cost and maintenance (Angehrn 1996).

A study coducted in **Tanzania** showed that vendors often dispensed smaller quantities of pesticides in unlabeled containers. About a third of the 61 small-scale farmers applied pesticides in mixtures. Up to 90% of this third had a maximum of 3 pesticides in a mixture. In all cases, there were no specific instructions either from the labels or extension workers regarding these tank mixtures. More than 50% of the respondents applied pesticides up to 5 times or more per cropping season depending on the crop. Insecticides and fungicides were routinely applied by 77% and 7%, respectively. Routine application implies regular, usually weekly and habitual application to prevent an anticipated pest attack. Sixty-eight percent of farmers reported having felt sick after routine application of pesticides. Pesticide-related health symptoms that were associated with pesticides use included skin problems and neurological system disturbances (dizziness, headache). Thirty-nine percent of farmers reported spending between 20 and 130,200 Tanzanian shillings (0.018–116 US dollars) in a year on health due to pesticides (Ngowi et al. 2007).

In **Burkina Faso**, 650 agricultural producers were surveyed in summer 2010. Among these farmers, 296 poisoning cases resulting from pesticide application operations were recorded. Overall, the study showed that farmers did not follow good agricultural practices and only about 1% wore appropriate personal protective equipment, which explains the high incidence of pesticide poisoning, and of acute cases as well, in a context where the medical care system is precarious and not easily accessible.

Farmers used very little personal protection during spraying. Some (25.9%), especially those within cooperatives, did use some

protective clothing. This included rubber boots, a coverall with long sleeves, gloves and a piece of cloth to cover the mouth. The majority wore trousers and a longsleeved shirt. However, some wore a short-sleeved shirt and short trousers, with no gloves, and barefooted farmers (they wore slippers which exposed a greater part of their feet) even used their bare hands to mix pesticides in a container. As a consequence, their legs, feet and hands came into contact with pesticides. About 80% of the farmers surveyed had become ill from pesticide exposure. The most frequent symptoms were reported as weakness, headache and/or dizziness. The commonest way of disposing of sprayer wash water (88.3%) and empty pesticide containers (80.2%) among the farmers interviewed was by throwing then on the field.

Asia

In a survey in **Cambodia**, 96% of interviewed farmers had experienced symptoms or signs of acute pesticide poisoning; 89% reported wearing a longsleeved shirt and long pants during spraying, 11% wore shorts, 61% wore no protective mask (the cotton masks in use may have a limited efficiency) and 79.2% wore no boots (CEDAC 2004). These figures indicate that partial protection does not stop acute poisoning.

Another survey in **Cambodia** reported that none of the ten farmers surveyed wore protective equipment and that the arms, back and feet of all ten farmers were soaked with pesticides after spraying (Yan et al 2001). A survey of 123 farmers in Thailand found that practically all wore a longsleeved shirt and long pants, 48% wore a mask made of cloth, 17% a sponge mask and 35% wore no mask; 105 of these farmers used paraquat (IPM Danida 2004). The signs and symptoms of poisoning that farmers reported were moderate in 63.4% of farmers (nausea, blurred vision, tremor, muscle cramps, chest pain or vomiting), mild in 34.1% (dry throat, dizziness, exhaustion, headache, shaky heart, itchy skin, weakness of muscles, skin rashes or sore throat), severe in 1.6% (convulsions or loss of consciousness), while only 0.8% of farmers had no symptoms (IPM Danida 2004).

In **Malaysia** a survey of 72 female plantation workers found that twothirds of them had been supplied with some protective equipment: 61.1% had received a respiratory mask, 44.4% gloves, 23.6% boots, 15.3% a cover for eyes and the face, 11% an overall, 1.4% an apron, while a third received no protective equipment. Few workers wore the mask as it was uncomfortable in the heat (Tenaganita & PANAP 2002).

In **Indonesia** it was found that farmers wore long (or knee-high) pants and a longsleeved shirt in less than half of spray operations (42% and 37%, respectively). Discomfort in the hot climate and the high cost of adequate protective clothing were the reasons. But skin and clothes were considerably contaminated by pesticide solutions and equipment was leaking in over half of the spray operations (Kishi et al. 1995).

Studies in **Thailand** on protective clothing for agricultural workers found that it was necessary to combine effective use of protective equipment with precautions for less hazardous handling and good personal hygiene (Chester et al 1990). But conditions in the field often do not allow this.

In **China**, (around 2000) pesticide poisoning caused about 4,000 deaths per year; an estimated 300 to 500 of these deaths were due to using pesticides in an «improper» manner (overuse, lack of protection) (Huang et al. 2000).

Among rice farmers in Zheijang, **China**, it was estimated that health costs from pesticide related illness were at least 15% of pesticide costs. They could be higher than the total cost of purchase if health costs for chronic diseases were included; about half of the poisoning cases were related to the use in agriculture (Huang et al. 2000).

A study in **China** found that the knapsack sprayers mostly in use were of inferior quality and leakages occurred frequently (Matthews 1996).

In **Pakistan**, a survey inclding 105 tobacco farmers showed that most of the farmers did not use any personal protective equipment during pesticide handling. Only a few used shoes (31%), masks (14%) and gloves (9%) during pesticide spray. In conclusion, the tobacco farmers had mild to moderate pesticide poisoning, which was correlated with depression in PChE (plasma cholinesterase) levels. Moreover, most farmers had little knowledge about the necessary safety measures, and displayed a casual attitude and unsatisfactory safety practices with regard to the use of basic protective equipment during pesticide applications on the tobacco crop (Khan et al.2010).

In the **Philippines**, of the 104 farmers interviewed, 31 reported using a knapsack sprayer that was currently leaking. At the time of the study, proper protective clothing such as rubber gloves, boots, a rubber apron and professional respiratory protection was not available to the farmers. The usual clothing worn during spraying consisted of thin trousers or shorts, a T-shirt or long-sleeved shirt, often with holes, bare feet or toe slippers, some form of headcover (mainly for protection from the sun rather than pesticides) and a cotton cloth tied in front of the mouth and nose to limit inhalation (Snelder et al. 2008).

A study on greenhouse workers (n=131) in **Turkey** showed that, none of the pesticide applicators used protective clothes, 88.5% of them stated that they even did not use protective gloves while they have applied pesticides. According to Turkish regulations, users are obliged to obtain certificates to use methly bromide, but 38 greenhouse workers stated that they use it without such certificates. This finding supports the idea of a general view that the existence of the legislation is not enough to enforce it (Ergonen et al. 2005).

Latin America/Caribbean

In **Nicaragua** it was estimated that 25% of workers experienced pesticide poisoning each year and 48% during their life (Keifer et al. 1996). A survey of agricultural workers in Yucatan, Mexico, found that in one year 40% had sought health care due to illness from exposure to pesticides (Drucker et al. 1999). Many workers on banana plantations use acutely toxic pesticides including paraquat without having received appropriate instructions (Foro Emaus 1998).

In **Brazil** a survey of spraying equipment found that all sprayers in use for over two years presented failures: the nozzle was in bad condition in 80.5% of sprayers, 56.6% had leaks and 47% had a damaged hose (Atuniassi & Gandolfi 2005). Another survey in the Amazonas area of **Brazil** showed that farmers do not use personal protective equipment and only 2% use gloves, protecting themselves only with a piece of cloth over the mouth and nose due to the belief that inhalation is the main route of toxic exposure, neglecting skin protection. Leftovers of pesticides are used to spray houses to eliminate insects or to spray facilities where food items like manioc flavor is produced and stored (Waichman et al. 2002).

USA

In California 13% of farm workers had no access to water, while symptoms reported at work were eye irritation (23% of workers), headache (15%), blurred vision (12%), skin irritation (12%), dizziness (5%), numbness or tingling (6%), nausea/vomiting (2.5%), diarrhoea (2%) and dehydration (1.5%) (CE 2000). Workers reentering sprayed fields may be highly exposed and even labour contractors often do not know what pesticide was sprayed (Bade 1999).

Inadequate working conditions prevail despite the responsibility of employers to be informed about safety requirements (in regulations and on product labels) and to inform workers about hazards and measures for protection (CDPR 2001). Among illness cases in California due to paraquat, the majority (39.1%) occurred during handling of spray equipment (by cleaning, due to a malfunction such as leakage or splashes during loading); one third of illnesses were due to various factors including 12.4% environmental causes (e.g. change of wind, spray drift), 11% accidents and 7.1% accidental contact with paraquat during the spraying or handling (Weinbaum et al. 1995).

The rate of paraquat-related illness cases associated with manual spraying was 18 times higher than with tractormounted sprayers. Other factors with a higher risk of illness were the crop type (e.g. fruit trees) and season the higher illness rates in summer may arise from less protective clothing being worn, increased paraquat absorption, and different physiological response at higher temperatures (Weinbaum et al. 1995).

Europe

A survey among 223 **Greek** tobacco farmers revealed that almost all farmers (99%) thought that pesticides can have serious adverse effects on users' health. Skin contact was recognized as the most common route of exposure during pesticide use (58%). Despite awareness of potential health risks by pesticide handling, a significant proportion of the farmers (46%) reported not using any special protective equipment when spraying pesticides. From those who reported that they use protective equipment, most stated that they normally use a hat (47%) and boots (63%). Only few farmers reported using a face mask (3%), gloves (8%), and coveralls (7%) on a regular basis. The reasons for not using protective equipment during pesticide handling were